Access DB# 62555

SEARCH REQUEST FORM

Scientific and Technical Information Center

Requester's Full Name: P. SKWIRK Examiner #: 70400 Date: 3/15/02
Art Unit: 1614 Phone Number 30 8 4703 Serial Number: 09/820,221
Mail Box and Bldg/Room Location: _____ Results Format Preferred (circle): PAPER DISK E-MAIL

If more than one search is submitted, please prioritize searches in order of need. 1 of 1

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Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

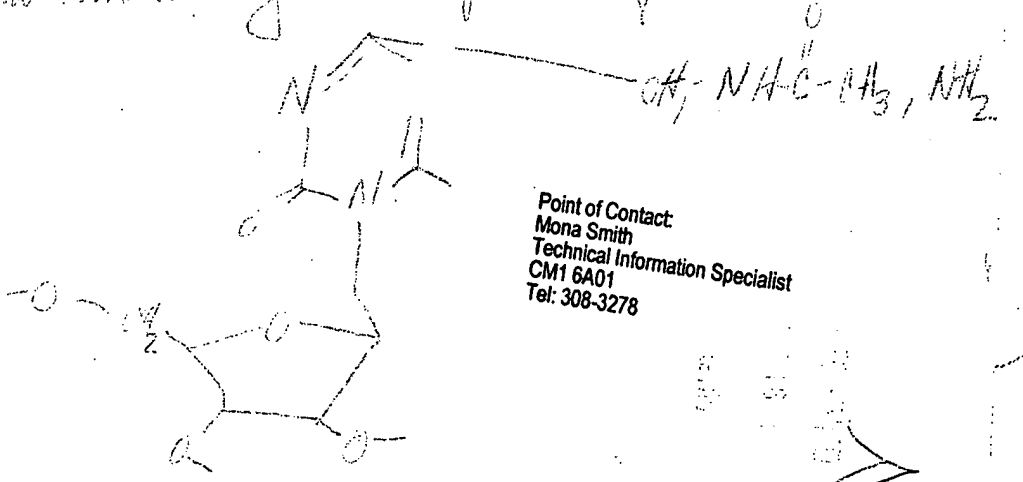
Title of Invention: Mitochondrial disorders

Inventors (please provide full names): Robert K. Naviaux

Earliest Priority Filing Date: 2/23/99

For Sequence Searches Only *Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.*

Please search methods of treating a mitochondrial disorder comprising administering a compound of



2',3',5'-tri-O-acetyl-1-β-D-uridine

Point of Contact:
Mona Smith
Technical Information Specialist
CM1 6A01
Tel: 308-3278

Thanks

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STAFF USE ONLY

Searcher: M. Smith
Searcher Phone #: _____
Searcher Location: _____
Date Searcher Picked Up: 3/17/02
Date Completed: 3/20/02
Searcher Prep & Review Time: 30
Clerical Prep Time: _____
Online Time: 30

Type of Search

NA Sequence (#) _____
AA Sequence (#) _____
Structure (#) X
Bibliographic _____
Litigation _____
Fulltext _____
Patent Family _____
Other _____

Vendors and cost where applicable

STN _____
Dialog _____
Questel/Orbit _____
Dr.Link _____
Lexis/Nexis _____
Sequence Systems _____
WWW/Internet _____
Other (specify) _____

PTO-1590 (8-01)

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=> fil hcaplu
FILE 'HCAPLUS' ENTERED AT 16:05:35 ON 20 MAR 2002
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FILE COVERS 1907 - 20 Mar 2002  VOL 136 ISS 12
FILE LAST UPDATED: 18 Mar 2002  (20020318/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

CAS roles have been modified effective December 16, 2001. Please
check your SDI profiles to see if they need to be revised. For
information on CAS roles, enter HELP ROLES at an arrow prompt or use
the CAS Roles thesaurus (/RL field) in this file.

The P indicator for Preparations was not generated for all of the
CAS Registry Numbers that were added to the CAS files between 12/27/01
and 1/23/02.  As of 1/23/02, the situation has been resolved.  Searches
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Numbers with the P indicator executed between 12/27/01 and 1/23/02 may
be incomplete.  See the NEWS message on this topic for more information.

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L1              STR
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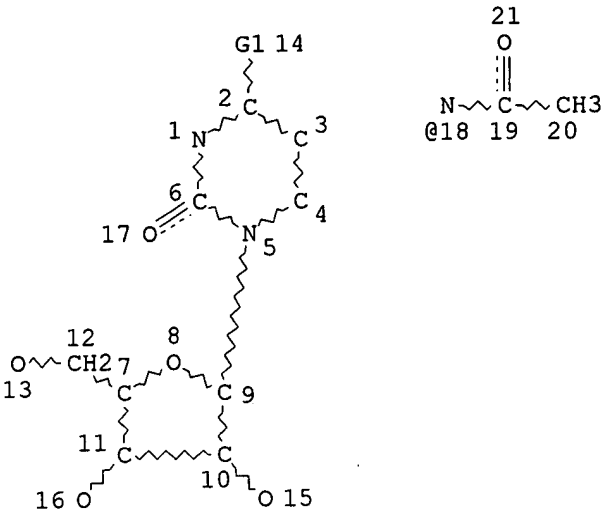


VAR G1=OH/18/NH2
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 21

STEREO ATTRIBUTES: NONE
L3 21230 SEA FILE=REGISTRY SSS FUL L1
L4 36336 SEA FILE=HCAPLUS L3
L6 295 SEA FILE=HCAPLUS L4 (L) (MITOCHOND? OR ATAXIA OR NEUROPATH? OR RETINITIS(W)PIGMENT? OR NARP (W)SYNDROME?)
L7 48 SEA FILE=HCAPLUS L6 AND (?MEDIC? OR ?DRUG? OR ?PHARM? OR ?THERAP?)

=> d ibib abs hitrn l7 1-48

L7 ANSWER 1 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:51460 HCAPLUS
DOCUMENT NUMBER: 136:112670
TITLE: Methods using purine derivatives, pyrimidine derivatives, and tetrahydroindolone derivatives for treatment of **drug**-induced peripheral neuropathy and related conditions
INVENTOR(S): Diamond, Jack; Glasky, Alvin J.
PATENT ASSIGNEE(S): Neotherapeutics, Inc., USA
SOURCE: PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002004448	A2	20020117	WO 2001-US21373	20010706

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-216844P P 20000707
OTHER SOURCE(S): MARPAT 136:112670

AB A method of treating **drug**-induced peripheral neuropathy comprises administering to a patient with **drug**-induced peripheral neuropathy an effective quantity of a purine deriv. or analog, a tetrahydroindolone deriv. or analog, or a pyrimidine deriv. or analog. If the compd. is a purine deriv., the purine moiety can be guanine or hypoxanthine. The compd. can induce peripheral nerve sprouting through the action of a neurotrophic factor such as nerve growth factor (NGF) without the occurrence of hyperalgesia. The peripheral nerve sprouting can be nociceptive nerve sprouting. The **drug**-induced peripheral neuropathy can be **drug**-induced peripheral neuropathy assocd. with the administration of oncolytic **drugs**, such as a vinca alkaloid, cisplatin, paclitaxel, suramin, altretamine, carboplatin, chlorambucil, cytarabine, dacarbazine, docetaxel, etoposide, fludarabine, ifosfamide with mesna, tamoxifen, teniposide, or thioguanine. The methods of the invention are particularly useful in treating peripheral neuropathy assocd. with the administration of vincristine, paclitaxel, or cisplatin.

IT **147-94-4**, Cytarabine
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(purine derivs., pyrimidine derivs., and tetrahydroindolone derivs. for treatment of **drug**-induced peripheral **neuropathy** and related conditions)

L7 ANSWER 2 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:727758 HCAPLUS
DOCUMENT NUMBER: 136:95674
TITLE: Bystander effects of nucleoside analogs phosphorylated in the cytosol or mitochondria
AUTHOR(S): Sanda, Alina; Zhu, Chaoyong; Johansson, Magnus; Karlsson, Anna
CORPORATE SOURCE: Karolinska Institute, Division of Clinical Virology, Huddinge University Hospital, Stockholm, S-141 86, Swed.
SOURCE: Biochemical and Biophysical Research Communications (2001), 287(5), 1163-1166
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic Press

DOCUMENT TYPE: Journal
LANGUAGE: English
AB The efficiency of nucleoside kinase suicide gene **therapy** for cancer is highly dependent on "bystander" cell killing, i.e., the transfer of cytotoxic phosphorylated nucleoside analogs to cells adjacent to those expressing the suicide enzyme. We have recently studied the possible use of mitochondrial nucleoside kinases as suicide genes. In the present study, we investigated if nucleoside analogs phosphorylated in the mitochondrial matrix cause bystander killing. We used deoxycytidine kinase-deficient Chinese hamster ovary cells reconstituted with deoxycytidine kinase targeted to either the cytosol or mitochondria matrix and detd. the bystander cell killing when these cells were incubated with the nucleoside analogs 1-.beta.-d-arabinofuranosylcytosine and 2',2'-difluorodeoxycytidine. A bystander effect occurred when nucleoside analogs were phosphorylated in the cytosol, but not when these compds. were phosphorylated in the mitochondria. These findings suggest that nucleoside kinases targeted to the mitochondrial matrix have limited use in suicide gene **therapy** when efficient bystander cell killing is required. (c) 2001 Academic Press.
IT **147-94-4**, AraC
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(bystander effects of nucleoside analogs phosphorylated in the cytosol or **mitochondria**)
REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:757703 HCAPLUS
DOCUMENT NUMBER: 134:51114
TITLE: Adriamycin-induced inhibition of mitochondrial-encoded polypeptides as a model system for the identification of hotspots for DNA-damaging agents
AUTHOR(S): Sharples, Robyn A.; Cullinane, Carleen; Phillips, Don R.
CORPORATE SOURCE: Department of Biochemistry, La Trobe University, Bundoora, 3083, Australia
SOURCE: Anti-Cancer Drug Design (2000), 15(3), 183-190
CODEN: ACDDEA; ISSN: 0266-9536
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB It has recently been shown that the anti-cancer **drug** Adriamycin forms **drug**-DNA adducts which function as "virtual" interstrand cross-links in cells, and these cross-links are specific for GpC sequences. The objective of this work was to det. whether all GpC sites are equally susceptible to the formation of Adriamycin-DNA adducts in the mitochondrial genome or whether any "hotspots" exist whereby lesions are formed preferentially at particular GpC-contg. sequences. The mitochondrial genome was used as a model system as it provides a series of contiguous genes, all of which lack introns and in which transcription is driven from a single promoter. With the absence of nucleotide excision repair, this provides an excellent system with which to observe Adriamycin-induced DNA damage since such lesions are reflected as an

inhibition of mitochondrial protein synthesis. HeLa cells were treated with Adriamycin and the extent to which synthesis of individual mitochondrial-encoded proteins was inhibited was quantitated. Mitochondrial protein synthesis was found to be inhibited in a discontinuous manner, corresponding to regions rich in 5'-GpC sequences. These results therefore indicate that Adriamycin-DNA adducts do not form randomly with GpC sites throughout the mitochondrial genome, but instead appear to form preferentially at regions of high GpC content. This selective inhibition of mitochondrial-encoded proteins demonstrates the potential of this method for the in situ detection of localized regions of binding by DNA-acting **drugs**.

IT **4785-04-0**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Adriamycin induced inhibition of **mitochondrial** encoded polypeptides as a model system for identification of hotspots for DNA-damaging agents)

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:701309 HCAPLUS
DOCUMENT NUMBER: 134:51102
TITLE: Potentiation of 1-.beta.-d-arabinofuranosylcytosine-mediated mitochondrial damage and apoptosis in human leukemia cells (U937) overexpressing Bcl-2 by the kinase inhibitor 7-hydroxystaurosporine (UCN-01)
AUTHOR(S): Tang, L.; Boise, L. H.; Dent, P.; Grant, S.
CORPORATE SOURCE: Medical College of Virginia, Department of Microbiology, Virginia Commonwealth University, Richmond, VA, USA
SOURCE: Biochemical Pharmacology (2000), 60(10), 1445-1456
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Antileukemic interactions between the nucleoside analog 1-.beta.-d-arabinofuranosylcytosine (ara-C) and the kinase inhibitor 7-hydroxystaurosporine (UCN-01) have been examd. in relation to Bcl-2 expression/phosphorylation, mitochondrial damage, caspase activation, and loss of clonogenic potential. Subsequent exposure of ara-C-pretreated U937 cells (1 .mu.M; 6 h) to UCN-01 (300 nM; 24 h) resulted in marked potentiation of pro-caspase-3 and -9 cleavage/activation, poly(ADP-ribose)polymerase degrdn., diminished mitochondrial membrane potential (.DELTA..psi.m), enhanced cytochrome c release, redn. in the S-phase fraction, and induction of classic apoptotic morphol. features. Enforced expression of full-length Bcl-2 significantly protected cells (at 24 h) from ara-C/UCN-01-induced caspase activation and apoptosis, but was ineffective in preventing loss of .DELTA..psi.m and cytochrome c release. Ectopic expression of a Bcl-2 N-terminal phosphorylation loop-deleted protein (Bcl-2.DELTA.32-80) was more potent than its full-length counterpart in blocking **drug**-induced loss of .DELTA..psi.m, caspase activation, and apoptotic morphol., but not cytochrome c release. Examn. of cells at later intervals revealed that ectopic expression of

Bcl-2 or Bcl-2.DELTA.32-80 could only delay, but not prevent, mitochondrial damage, caspase activation, and cell death induced by ara-C/UCN-01 treatment. Despite their initial ability to inhibit apoptosis, neither full-length nor truncated Bcl-2 protein restored clonogenic potential to **drug**-treated cells. These findings indicate that subsequent exposure of ara-C-pretreated human leukemia cells to UCN-01 potently triggers mitochondrial damage and apoptosis, and that these events are postponed but not prevented by ectopic expression of Bcl-2 or its phosphorylation loop-deleted counterpart.

IT **147-94-4**, Ara-C
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(potentiation of ara-C-mediated **mitochondrial** damage and apoptosis in human leukemia cells (U937) overexpressing Bcl-2 by kinase inhibitor 7-hydroxystaurosporine)

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 5 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:641866 HCAPLUS
DOCUMENT NUMBER: 133:344310
TITLE: Incorporation of nucleoside analogs into nuclear or mitochondrial DNA is determined by the intracellular phosphorylation site
AUTHOR(S): Zhu, Chaoyong; Johansson, Magnus; Karlsson, Anna
CORPORATE SOURCE: Division of Clinical Virology, Karolinska Institute, Huddinge University Hospital, Stockholm, S-141 86, Swed.
SOURCE: Journal of Biological Chemistry (2000), 275(35), 26727-26731
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Nucleoside analogs used in cancer **chemotherapy** and in treatment of virus infections are phosphorylated in cells by nucleoside and nucleotide kinases to their **pharmacol**. active form. The phosphorylated nucleoside analogs are incorporated into DNA and cause cell death or inhibit viral replication. Cellular DNA is replicated both in the nucleus and in the mitochondria, and nucleoside analogs may interfere with DNA replication in both these subcellular locations. In the present study we created a cell model system where nucleoside analogs were phosphorylated, and thereby **pharmacol**. activated, in either the nucleus, cytosol, or mitochondria of cancer cells. The system was based on the reconstitution of deoxycytidine kinase (dCK)-deficient Chinese hamster ovary cells with genetically engineered dCK targeted to the different subcellular compartments. The nucleoside analogs phosphorylated by dCK in the mitochondria were predominantly incorporated into mitochondrial DNA, whereas the nucleoside analogs phosphorylated in the nucleus or cytosol were incorporated into nuclear DNA. We further show that the nucleoside analogs phosphorylated in the mitochondria induced cell death by an apoptotic program. These data showed that the

subcellular site of nucleoside analog phosphorylation is an important determinant for incorporation of nucleoside analogs into nuclear or mitochondrial DNA.

IT **147-94-4**, AraC
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(incorporation of nucleoside analogs into nuclear or **mitochondrial** DNA is detd. by the intracellular phosphorylation site and relevance to antitumor activity)

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 6 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:608584 HCAPLUS
DOCUMENT NUMBER: 133:187987
TITLE: Methods using pyrimidine-based nucleosides for treatment of mitochondrial disorders
INVENTOR(S): Naviaux, Robert K.
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000050043	A1	20000831	WO 2000-US4663	20000223
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1171137	A1	20020116	EP 2000-910321	20000223
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1999-121588P P 19990223
WO 2000-US4663 W 20000223

OTHER SOURCE(S): MARPAT 133:187987

AB Methods are provided for the treatment of mitochondrial disorders. The methods include the administration of a pyrimidine-based nucleoside, e.g. triacetyluridine. Also provided are methods of reducing or eliminating symptoms assocd. with mitochondrial disorders. Mitochondrial disorders particularly appropriate for treatment include those attributable to a deficiency of one or more pyrimidines.

IT **4105-38-8**
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pyrimidine-based nucleoside for treatment of **mitochondrial**

disorder)
IT **58-96-8**, Uridine
RL: BPR (Biological process); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(pyrimidine-based nucleoside for treatment of **mitochondrial** disorder)
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 7 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:380966 HCAPLUS
DOCUMENT NUMBER: 134:142417
TITLE: Transcriptional and post-transcriptional in organello labelling of Trypanosoma brucei mitochondrial RNA
AUTHOR(S): Militello, K. T.; Hayman, M. L.; Read, L. K.
CORPORATE SOURCE: Department of Microbiology and Center for Microbial Pathogenesis, SUNY at Buffalo School of Medicine, Buffalo, NY, 14214, USA
SOURCE: International Journal for Parasitology (2000), 30(5), 643-647
CODEN: IJPYBT; ISSN: 0020-7519
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In organello labeling of Trypanosoma brucei mitochondrial (mt) RNA was characterized with respect to nucleotide requirements and **drug** sensitivity. Mitochondrial transcriptional activity is maximal in the presence of all ribonucleoside-triphosphate NTPs, and can be inhibited by UTP depletion. Mitochondrial transcription can also be partially inhibited by actinomycin D (actD) or ethidium bromide (EtBr). Post-transcriptional UTP incorporation is insensitive to actinomycin D or ethidium bromide. Proteins were identified that interact with transcriptional and post-transcriptionally labeled RNAs, and confirm the in vitro RNA-binding properties discovered for a no. of T. brucei mt proteins. These expts. reveal new strategies for studying mt transcription and processing in T. brucei mitochondria.
IT **63-39-8**, UTP
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(transcriptional and post-transcriptional in organello labeling of Trypanosoma brucei **mitochondrial** RNA in response to UTP depletion)
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 8 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:364242 HCAPLUS
DOCUMENT NUMBER: 133:129607
TITLE: Differential incorporation of 1-.beta.-D-arabinofuranosylcytosine and 9-.beta.-D-arabinofuranosylguanine into nuclear and mitochondrial DNA
AUTHOR(S): Zhu, C.; Johansson, M.; Karlsson, A.
CORPORATE SOURCE: Division of Clinical Virology, Karolinska Institute,

Huddinge University Hospital, Stockholm, S-141 86, Swed.
SOURCE: FEBS Lett. (2000), 474(2,3), 129-132
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The anti-leukemic nucleoside analogs 1-.beta.-D-arabinofuranosylcytosine (araC) and 9-.beta.-D-arabinofuranosylguanine (araG) are dependent on intracellular phosphorylation for **pharmacol**. activity. AraC is efficiently phosphorylated by deoxycytidine kinase (dCK). Although araG is phosphorylated by dCK in vitro, it is a preferred substrate of mitochondrial deoxyguanosine kinase. We have used autoradiog. to show that araC was incorporated into nuclear DNA in Molt-4 and CEM T-lymphoblastoid cells as well as in Chinese hamster ovary cells. In contrast, araG was predominantly incorporated into mitochondrial DNA in the investigated cell lines, without detectable incorporation into nuclear DNA. These data suggest that the mol. targets of araG and araC may differ.
IT **147-94-4**, 1-.beta.-D-Arabinofuranosylcytosine
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(differential incorporation of araC and araG into nuclear and **mitochondrial** DNA)
REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 9 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:285481 HCAPLUS
DOCUMENT NUMBER: 133:246786
TITLE: Expression of human mitochondrial thymidine kinase in Escherichia coli: correlation between the enzymatic activity of pyrimidine nucleoside analogues and their inhibitory effect on bacterial growth
AUTHOR(S): Wang, J.; Su, C.; Neuhard, J.; Eriksson, S.
CORPORATE SOURCE: Department of Veterinary Medical Chemistry, Swedish University of Agricultural Sciences, The Biomedical Center, Uppsala, S-751 23, Swed.
SOURCE: Biochemical Pharmacology (2000), 59(12), 1583-1588
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mitochondrial thymidine kinase (TK2) phosphorylates pyrimidine nucleosides to monophosphates and is expressed constitutively through the cell cycle in all cells. Because of the overlap of its substrate specificity with that of the cytosolic thymidine kinase (TK1) and deoxycytidine kinase (dCK), it has been difficult to det. the role of TK2 in activating nucleosides used in **chemotherapy**. In this report, we described the construction of a recombinant Escherichia coli strain which could be used to test if TK2 activity is limiting for the toxicity of nucleosides. Enzymes of bacterial origin which are involved in thymidine and deoxyuridine anabolism and catabolism were eliminated, and the cDNA for

human TK2 was introduced. In the crude ext. of the engineered E. coli, the level of thymidine kinase was, after induction of TK2 expression, several hundred fold higher than in the control strain. Several **pharmacol**. interesting nucleoside analogs, including 3'-azidothymidine, 2',3'-didehydro-2',3'-dideoxythymidine, and 2',3'-dideoxy-.beta.-l-3'-thiacytidine, were tested for their effects on the growth of this recombinant strain. For a comparison, the phosphorylation of these compds. was detd. with purified recombinant TK1, TK2, and dCK. A correlation was obsd. between the phosphorylation of several of these compds. by TK2 and their effects on bacterial growth. These results demonstrate that activation of growth-inhibiting pyrimidine nucleosides can be catalyzed by TK2, and together with recombinant E. coli strains expressing other cellular nucleoside kinases, this whole-cell bacterial system may serve as a tool to predict the efficacy and side effects of **chemotherapeutic** nucleosides.

IT **147-94-4**, 1-.beta.-D-Arabinofuranosylcytosine
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(AraC, pyrimidine nucleoside analog; phosphorylation of pyrimidine nucleoside analogs by human **mitochondrial** thymidine kinase (TK2) in E. coli and effect on growth)

IT **605-23-2**, 1-.beta.-D-Arabinofuranosylthymine
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(AraT, pyrimidine nucleoside analog; phosphorylation of pyrimidine nucleoside analogs by human **mitochondrial** thymidine kinase (TK2) in E. coli and effect on growth)

IT **3083-77-0**, 1-.beta.-D-Arabinofuranosyluracil
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(AraU, pyrimidine nucleoside analog; phosphorylation of pyrimidine nucleoside analogs by human **mitochondrial** thymidine kinase (TK2) in E. coli and effect on growth)

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 10 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:161074 HCAPLUS
DOCUMENT NUMBER: 132:203149
TITLE: Compositions and methods using pyrimidine nucleotide precursors for treatment of mitochondrial diseases
INVENTOR(S): Von Borstel, Reid W.
PATENT ASSIGNEE(S): Pro-Neuron, Inc., USA
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2000011952	A1	20000309	WO 1999-US19725	19990831
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2001005719	A1	20010628	US 1998-144096	19980831
AU 9960219	A1	20000321	AU 1999-60219	19990831
BR 9913319	A	20010522	BR 1999-13319	19990831
EP 1109453	A1	20010627	EP 1999-968207	19990831
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2001016576	A1	20010823	US 2001-838136	20010420

PRIORITY APPLN. INFO.: US 1998-144096 A2 19980831
WO 1999-US19725 W 19990831

AB Compds., compns., and methods are provided for treatment of disorders related to mitochondrial dysfunction. The methods comprise administering to a mammal a compn. contg. pyrimidine nucleotide precursors in amts. sufficient to treat symptoms resulting from mitochondrial respiratory chain deficiencies.

IT **58-96-8**, Uridine
RL: BAC (Biological activity or effector, except adverse); RCT (Reactant); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pyrimidine nucleotide precursors for treatment of **mitochondrial** diseases)

IT **260360-01-8P**
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(pyrimidine nucleotide precursors for treatment of **mitochondrial** diseases)

IT **58-96-8D**, Uridine, acyl derivs. **65-46-3**, Cytidine **65-46-3D**, Cytidine, acyl derivs. **987-78-0**, Cytidine diphosphocholine **4105-38-8**, 2',3',5'-Tri-O-acetyluridine **260360-02-9 260360-03-0 260360-04-1 260360-05-2 260360-06-3 260360-07-4**
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pyrimidine nucleotide precursors for treatment of **mitochondrial** diseases)

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 11 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:140574 HCAPLUS
DOCUMENT NUMBER: 132:203163
TITLE: Antisense inhibition of mitochondrial phosphoenolpyruvate carboxykinase (PEPCK-mitochondrial) expression

INVENTOR(S): McKay, Robert; Butler, Madeline M.; Cowsert, Lex M.
PATENT ASSIGNEE(S): Isis Pharmaceuticals Inc., USA
SOURCE: U.S., 32 pp.
CODEN: USXXAM

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6030837	A	20000229	US 1999-366257	19990803
WO 2001009379	A1	20010208	WO 1999-US30660	19991223

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-366257 A 19990803

AB Antisense compds., compns. and methods are provided for modulating the expression of PEPCK-mitochondrial. The compns. comprise antisense compds., particularly antisense oligonucleotides, targeted to nucleic acids encoding PEPCK-mitochondrial. Methods of using these compds. for modulation of PEPCK-mitochondrial expression and for treatment of diseases assocd. with expression of PEPCK-mitochondrial are provided.

IT **212061-30-8P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(antisense inhibition of **mitochondrial** phosphoenolpyruvate carboxykinase expression)

IT **163759-49-7P 163759-50-0P 182495-98-3P 182496-00-0P 212061-24-0P 212061-25-1P 212061-27-3P 212061-28-4P 212061-29-5P 244277-62-1P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction; antisense inhibition of **mitochondrial** phosphoenolpyruvate carboxykinase expression)

IT **1463-10-1**, 5-Methyluridine
RL: RCT (Reactant)
(reaction; antisense inhibition of **mitochondrial** phosphoenolpyruvate carboxykinase expression)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 12 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:41148 HCAPLUS
DOCUMENT NUMBER: 132:165037
TITLE: CpG DNA rescues B cells from apoptosis by activating NF.kappa.B and preventing mitochondrial membrane potential disruption via a chloroquine-sensitive pathway

AUTHOR(S): Yi, Ae-Kyung; Peckham, Dave W.; Ashman, Robert F.; Krieg, Arthur M.
CORPORATE SOURCE: Interdisciplinary Graduate Program in Immunology and Department of Internal Medicine, University of Iowa College of Medicine, Iowa City, IA, 52242, USA
SOURCE: Int. Immunol. (1999), 11(12), 2015-2024
CODEN: INIMEN; ISSN: 0953-8178
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Isolated murine splenic B cells gradually undergo spontaneous apoptosis while WEHI-231 B lymphoma cells undergo activation-induced apoptosis. Unmethylated CpG dinucleotides in a particular sequence context (CpG motif) in bacterial DNA or in synthetic oligodeoxynucleotides (CpG DNA) rescue both splenic B cells and WEHI-231 cells from apoptosis, an effect which could potentially contribute to autoimmune disease. Chloroquine has been used as an effective **therapeutic** agent for some autoimmune diseases, although the mechanism of action is not clearly understood. Low concns. of chloroquine (<5 .mu.M) selectively abolished CpG DNA-mediated protection against spontaneous apoptosis of splenic B cells and against anti-IgM-induced apoptosis of WEHI-231 cells without affecting anti-apoptotic activities of anti-CD40 or lipopolysaccharide. CpG DNA effectively prevented mitochondrial membrane potential disruption through a chloroquine-sensitive pathway in splenic B cells. Apoptosis protection by CpG DNA was also assocd. with increased expression of several proto-oncogenes and oncoproteins directly and/or indirectly through a rapid and sustained activation of NF.kappa.B in splenic B cells and WEHI-231 cells. These effects were also suppressed by chloroquine. Our results suggest that despite the difference in maturation phenotype of splenic B cells and WEHI-231 cells, CpG DNA rescues both from apoptosis by similar pathway, which is blocked at an early step by chloroquine.

IT **2382-65-2**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BIOL (Biological study); PROC (Process)
(CpG DNA rescues B cells from apoptosis by activating NF.kappa.B and preventing **mitochondrial** membrane disruption via a chloroquine-sensitive pathway)

REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 13 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:690981 HCAPLUS
DOCUMENT NUMBER: 131:327494
TITLE: Regulating cell proliferation by regulating mitochondrial metabolism and expression of cell-surface immunoproteins
INVENTOR(S): Newell, Martha K.
PATENT ASSIGNEE(S): University of Vermont, USA
SOURCE: PCT Int. Appl., 124 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9953953	A2	19991028	WO 1999-US6874	19990330
WO 9953953	A3	20000113		

W: AU, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

AU 9933705	A1	19991108	AU 1999-33705	19990330
EP 1077724	A2	20010228	EP 1999-915109	19990330

R: DE, DK, ES, GB, SE, PT

PRIORITY APPLN. INFO.:			
	US 1998-82250P	P	19980417
	US 1998-94519P	P	19980729
	US 1998-101580P	P	19980924
	WO 1999-US6874	W	19990330

AB The invention involves methods of regulating cell growth and division to control disease processes by manipulating mitochondrial metab. and the expression of cell surface immune proteins. The invention also involves related compns. and screening assays.

IT **147-94-4**, Cytarabine
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(regulating cell proliferation by regulating **mitochondrial** metab. and expression of cell-surface immunoproteins)

L7 ANSWER 14 OF 48 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER:	1999:673008 HCAPLUS
DOCUMENT NUMBER:	132:18546
TITLE:	Loss of mitochondrial membrane potential is dependent on the apoptotic program activated: prevention by R-2HMP
AUTHOR(S):	Zhang, D.; Berry, M. D.; Paterson, I. A.; Boulton, A. A.
CORPORATE SOURCE:	Neuropsychiatry Research Unit, Department of Psychiatry, University of Saskatchewan, Saskatoon, SK, S7N 5E4, Can.
SOURCE:	J. Neurosci. Res. (1999), 58(2), 284-292 CODEN: JNREDK; ISSN: 0360-4012
PUBLISHER:	Wiley-Liss, Inc.
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB Recent evidence suggests that the mitochondrial membrane potential begins to decrease well before the cells commit to apoptotic death. By using cultured cerebellar granule cells, two types of apoptosis can be induced, one by adding cytosine arabinoside (Ara-c; p53-dependent apoptosis) and one by lowering the K+ concns. of the medium (p53-independent apoptosis). Cultures show clear signs of increased apoptosis (chromatin condensation as visualized with bisbenzamide) after 12 h which increases with time up to 24 h. A fluorescent probe, chloromethyl-tetramethylrhodamine Me ester (CMTMR), a lipophilic, potentiometric dye, which when introduced into the media accumulates within mitochondria in proportion to the mitochondrial membrane potential, was added at various time points after the induction of apoptosis. In Ara-c-induced apoptosis, there was a shift in the distribution of cell populations towards low-intensity CMTMR fluorescence,

whereas in control and low-K+ cultures, there was no such shift. This effect was obsd. as early as 6 h after adding Ara-c. The antiapoptotic **drug** R-N-2-heptyl-N-methylpropargylamine hydrochloride (R-2HMP) reversed this loss of mitochondrial membrane potential in Ara-c-induced apoptosis; the effect was antagonized by the S-2HMP.

IT **147-94-4**, Ara-c
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(loss of **mitochondrial** membrane potential is dependent on the apoptotic program activated: prevention by R-2HMP)

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 15 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:453397 HCAPLUS
DOCUMENT NUMBER: 131:281217
TITLE: Deletions in the mitochondrial DNA and decrease in the oxidative phosphorylation activity of children with Fanconi syndrome secondary to antiblastic **therapy**
AUTHOR(S): Di Cataldo, Andrea; Palumbo, Maddalena; Pittala, Donatella; Renis, Marcella; Schiliro, Gino; Russo, Alessandra; Ragusa, Rosalia; Mollica, Florindo; Li Volti, Salvatore
CORPORATE SOURCE: Departments of Pediatric Hematology-Oncology, Biochemistry, and Pediatrics, University of Catania, Italy
SOURCE: Am. J. Kidney Dis. (1999), 34(1), 98-106
CODEN: AJKDDP; ISSN: 0272-6386
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The aim of this study is to verify whether there are deletions in mitochondrial DNA (mtDNA) and disorders in oxidative phosphorylation (Ox-phos) complexes in the pathogenesis of secondary Fanconi syndrome (FS). The authors studied 18 children with tumors who were previously treated with **chemotherapy** and were off **therapy** for at least 1 yr. All the children had normal renal function at diagnosis. Only 4 children received ifosfamide (IFO) and platinum compds. The authors evaluated renal function, Ox-phos activity measured on platelets, and mtDNA extd. from platelets for all patients. Only 2 patients, both treated with IFO and carboplatinum (CARBO) for Wilms' tumor and germ-cell tumor, resp., developed FS 1 and 3 yr after termination of **therapy**. They had decreased activities of Ox-phos that were statistically significant only for NAD-reduced cytochrome-c reductase and cytochrome-c oxidase and specific and unidentified deletions in mtDNA that were not maternally inherited. Therefore, treatment with IFO and CARBO might be responsible for deletions in mtDNA, decreased activity of Ox-phos, and impaired rates of transport of D-glucose, phosphate, and amino acids.

IT **147-94-4**, Cytarabine
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(deletions in **mitochondrial** DNA and decreases in oxidative phosphorylation activity in children with Fanconi syndrome secondary to

antiblastic **therapy**)
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 16 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:27861 HCAPLUS
DOCUMENT NUMBER: 130:80354
TITLE: Methods and compositions for galactosylated glycoproteins
INVENTOR(S): Raju, Shantha T.
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9858964	A1	19981230	WO 1998-US13066	19980623
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9881634	A1	19990104	AU 1998-81634	19980623
EP 994903	A1	20000426	EP 1998-931522	19980623
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002506353	T2	20020226	JP 1999-504999	19980623

PRIORITY APPLN. INFO.: US 1997-881301 A 19970624
WO 1998-US13066 W 19980623

AB This invention relates to novel glycoprotein glycoform prepns. comprising the substantially homogeneous glycoprotein glycoforms. The glycoprotein is antibody, monoclonal antibody, IgG, IgG1, or immunoadhesin, e.g. anti-CD20, anti-HER2, anti-VEGF, anti-IgE, and anti-TNF receptor antibody. More particularly the invention relates to substantially homogeneous glycoprotein prepns. comprising a particular Fc glycan and methods for producing, detecting, enriching and purifying the glycoforms. The invention further relates to Igs and esp. antibodies comprising a CH2 domain having a particular glycan. Provided are compns. including **pharmaceutical** compns., methods of using the prepns. as well as articles of manuf. comprising the prepns. The compns. are prepd. by treating substrate glycoprotein with metal salt, activated galactose and galactosyltransferase. The compns. are useful for treating inflammatory disorder, cancer neurofibromatosis, peripheral neuropathologies and cardiac hypertrophy.

IT **2956-16-3**, UDP-galactose
RL: BUU (Biological use, unclassified); RCT (Reactant); BIOL (Biological study); USES (Uses)

(galactosylated glycoproteins such as Ig. and antibody and immunoadhesin for treating neurofibromatosis, peripheral **neuropathologies** and cardiac hypertrophy)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 17 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:645601 HCAPLUS
DOCUMENT NUMBER: 130:255
TITLE: A functional role for mitochondrial protein kinase C.alpha. in Bcl2 phosphorylation and suppression of apoptosis
AUTHOR(S): Ruvolo, Peter P.; Deng, Xingming; Carr, Boyd K.; May, W. Stratford
CORPORATE SOURCE: Sealy Center for Oncology and Hematology and the Department of Internal Medicine, University of Texas Medical Branch, Galveston, TX, 77555, USA
SOURCE: J. Biol. Chem. (1998), 273(39), 25436-25442
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Phosphorylation of Bcl2 at serine 70 may result from activation of a classic protein kinase C (PKC) isoform and is required for functional suppression of apoptosis by Bcl2 in murine growth factor-dependent cell lines. Human pre-B REH cells express high levels of Bcl2 yet remain sensitive to the **chemotherapeutic** agents etoposide, cytosine arabinoside, and Adriamycin. In contrast, myeloid leukemia-derived HL60 cells express less than half the level of Bcl-2 but are >10-fold more resistant to apoptosis induced by these **drugs**. The mechanism responsible for this apparent dichotomy appears to involve a deficiency of mitochondrial PKC.alpha. since 1) HL60 but not REH cells contain highly phosphorylated Bcl2; 2) PKC.alpha. is the only classical isoform co-localized with Bcl2 in HL60 but not REH mitochondrial membranes; 3) the natural product and potent PKC activator bryostatin-1 induces mitochondrial localization of PKC.alpha. in assocn. with Bcl2 phosphorylation and increased REH cell resistance to **drug** -induced apoptosis; 4) PKC.alpha. can directly phosphorylate wild-type but not phosphorylation-neg. and loss of function S70A Bcl2 in vitro; 5) stable, forced expression of exogenous PKC.alpha. induces mitochondrial localization of PKC.alpha., increased Bcl2 phosphorylation and a >10-fold increase in resistance to **drug**-induced cell death; and (6) PKC.alpha.-transduced cells remain highly sensitive to staurosporine, a potent PKC inhibitor. Furthermore, treatment of the PKC.alpha. transformants with bryostatin-1 leads to even higher levels of mitochondrial PKC.alpha., Bcl2 phosphorylation, and REH cell survival following **chemotherapy**. While these findings strongly support a role for PKC.alpha. as a functional Bcl2 kinase that can enhance cell resistance to antileukemic **chemotherapy**, they do not exclude the possibility that another Bcl2 kinase(s) may also exist. Collectively, these findings identify a functional role for PKC.alpha. in Bcl2 phosphorylation and in resistance to **chemotherapy** and suggest a novel target for antileukemic strategies.

IT **147-94-4**, Cytosine arabinoside
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(functional role for **mitochondrial** protein kinase C.alpha. in Bcl2 phosphorylation and suppression of apoptosis)
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 18 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:429146 HCAPLUS
DOCUMENT NUMBER: 129:170186
TITLE: Abrogation of mitochondrial cytochrome c release and caspase-3 activation in acquired **multidrug** resistance
AUTHOR(S): Kojima, Hiromi; Endo, Kazuya; Moriyama, Hiroshi; Tanaka, Yasuhiro; Alnemri, Emad S.; Slapak, Christopher A.; Teicher, Beverly; Kufe, Donald; Datta, Rakesh
CORPORATE SOURCE: Cancer Pharmacology, Dana-Faber Cancer Institute, Harvard Medical School, Boston, MA, 02115, USA
SOURCE: J. Biol. Chem. (1998), 273(27), 16647-16650
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Acquired **multidrug** resistance to anti-cancer agents has been assocd. with overexpression of the P-glycoprotein and other members of the ATP-binding cassette superfamily. The present studies demonstrate that SCC-25 cells selected for resistance to the alkylating agent cisplatin (CDDP) overexpress the anti-apoptotic Bcl-xL protein. In contrast to parental cells, the SCC-25/CDDP-resistant variant failed to exhibit activation of caspase-3, cleavage of protein kinase C .delta., and other characteristics of apoptosis in response to CDDP. Similar results were obtained when SCC-25/CDDP cells were exposed to the structurally and functionally unrelated antimetabolite 1-.beta.-D-arabinofuranosylcytosine (ara-C). Other cells selected for resistance to doxorubicin or vincristine also exhibited overexpression of Bcl-xL and failed to respond to CDDP and ara-C with activation of caspase-3. The results further demonstrate that **multidrug**-resistant cells exhibit a block in the release of mitochondrial cytochrome c into the cytosol and that this effect is dependent on overexpression of Bcl-xL. The demonstration that lysates from the resistant cells respond to the addn. of cytochrome c with activation of caspase-3 confirms that the block in apoptosis is because of inhibition of mitochondrial cytochrome c release. These findings demonstrate that cells respond to diverse classes of anti-cancer **drugs** with overexpression of Bcl-xL and that this response represents another mechanism of acquired **multidrug** resistance.
IT **147-94-4**, 1-.beta.-D-Arabinofuranosylcytosine
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(abrogation of **mitochondrial** cytochrome c release and caspase-3 activation in acquired **multidrug** resistance)

L7 ANSWER 19 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:129343 HCAPLUS
DOCUMENT NUMBER: 128:252614
TITLE: Bcr-Abl exerts its antiapoptotic effect against diverse apoptotic stimuli through blockage of mitochondrial release of cytochrome C and activation of caspase-3
AUTHOR(S): Amarante-Mendes, Gustavo P.; Kim, Caryn Naekyung; Liu, Linda; Huang, Yue; Perkins, Charles L.; Green, Douglas R.; Bhalla, Kapil
CORPORATE SOURCE: Division of Hematology/Oncology, Department of Medicine, Winship Cancer Center, Emory University School of Medicine, Atlanta, GA, 30322, USA
SOURCE: Blood (1998), 91(5), 1700-1705
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Bcr-Abl expression in leukemic cells is known to exert a potent effect against apoptosis due to antileukemic **drugs**, but its mechanism has not been elucidated. Recent reports have indicated that a variety of apoptotic stimuli cause the preapoptotic mitochondrial release of cytochrome c (cyt c) into cytosol, which mediates the cleavage and activity of caspase-3 involved in the execution of apoptosis. Whether Bcr-Abl exerts its antiapoptotic effect upstream to the cleavage and activation of caspase-3 or acts downstream by blocking the ensuing degrdn. of substrates resulting in apoptosis, has been the focus of the present studies. In these, the authors used (1) the human acute myelogenous leukemia (AML) HL-60 cells that are stably transfected with the bcr-abl gene (HL-60/Bcr-Abl) and express p185 Bcr-Abl; and (2) the chronic myelogenous leukemia (CML)-blast crisis K562 cells, which have endogenous expression of p210 Bcr-Abl. Exposure of the control AML HL-60 cells to high-dose Ara-C (HIDAC), etoposide, or sphingoid bases (including C2 ceramide, sphingosine, or sphinganine) caused the accumulation of cyt c in the cytosol, loss of mitochondrial membrane potential (MMP), and increase in the reactive oxygen species (ROS). These preapoptotic events were assocd. with the cleavage and activity of caspase-3, resulting in the degrdn. of poly (ADP [ADP]-ribose) polymerase (PARP) and DNA fragmentation factor (DFF), internucleosomal DNA fragmentation, and morphol. features of apoptosis. In contrast, in HL-60/Bcr-Abl and K562 cells, these apoptotic stimuli failed to cause the cytosolic accumulation of cyt c and other assocd. mitochondrial perturbations, as well as the failure to induce the activation of caspase-3 and apoptosis. While the control HL-60 cells showed high levels of Bcl-2 and barely detectable Bcl-xL, HL-60/Bcr-Abl cells expressed high levels of Bcl-xL and undetectable levels of Bcl-2, a pattern of expression similar to the one in K562 cells. Bax and caspase-3 expressions were not significantly different between HL-60/Bcr-Abl or K562 vs. HL-60 cells. These findings indicate that Bcr-Abl expression blocks apoptosis due to diverse apoptotic stimuli upstream by preventing the cytosolic accumulation of cyt c and other preapoptotic mitochondrial perturbations, thereby inhibiting the activation of caspase-3 and execution of apoptosis.

IT **147-94-4**, Ara C
RL: BAC (Biological activity or effector, except adverse); THU

(Therapeutic use); BIOL (Biological study); USES (Uses)
(Bcr-Abl exerts antiapoptotic effect through blockage of **mitochondrial** release of cytochrome C and activation of caspase-3 in relation to resistance to antileukemia **drugs**)

L7 ANSWER 20 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:679102 HCAPLUS
DOCUMENT NUMBER: 127:328394
TITLE: Nuclear and mitochondrial human dUTPase isoforms and their value as proliferation or tumor markers
INVENTOR(S): Ladner, Robert D.; Lynch, Frank; Caradonna, Salvatore J.
PATENT ASSIGNEE(S): University of Medicine and Dentistry of New Jersey, USA
SOURCE: PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9736916	A1	19971009	WO 1997-US4886	19970326
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5962246	A	19991005	US 1997-824405	19970326

PRIORITY APPLN. INFO.: US 1996-14748P P 19960329
US 1997-824405 A 19970326

AB DNA and amino acid sequences are provided for dUTPase, an enzyme which is essential for life and which is increased during periods of cellular proliferation. The human form of the dUTPase enzyme has 2 isoforms, a nuclear and a mitochondrial isoform, which have identical cDNA sequences in their overlapping regions. Characterization of the 5' region of the gene encoding dUTPase demonstrates that the dUTPase isoforms are encoded by the same gene with isoform-specific transcripts arising through the use of alternative 5' exons. The nuclear isoform (DUT-N) is a proliferation marker, and certain non-proliferating neoplasms have increased levels of cytoplasmic mitochondrial dUTPase (DUT-M) and are Ki-67 neg. Methods of detg. the proliferation status of a cell, efficacy of antineoplastic compds., and response to **therapy** with antineoplastic compds., using cellular levels of dUTPase is disclosed. The dUTPase proliferation marker method offers several advantages: (1) unlike Ki-67, dUTPase is essential to cell viability; (2) DUTPase is a stable enzyme, whereas ki-67 is rapidly degraded after cell death; (3) Ki-67 prodn. is turned off in nutritionally deprived cells, but this does not occur with dUTPase; (4) certain tumors which test neg. for proliferation with Ki-67 will test pos. for dUTPase; and (5) the 2 isoforms of dUTPase are readily distinguishable from each other. A kit contg. the necessary reagents for the detn. of dUTPase is also disclosed.

IT **147-94-4**, Cytosine arabinoside
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(assessment of efficacy of; nuclear and **mitochondrial** human dUTPase isoforms and their value as proliferation or tumor markers)

L7 ANSWER 21 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:396172 HCAPLUS
DOCUMENT NUMBER: 127:90225
TITLE: Relationships between the mitochondrial permeability transition and oxidative stress during ara-C toxicity
AUTHOR(S): Backway, Karen L.; Mcculloch, Ernest A.; Chow, Sue; Hedley, David W.
CORPORATE SOURCE: Department of Pathology, Ontario Cancer Institute/Princess Margaret Hospital, Toronto, ON, M5G 2M9, Can.
SOURCE: Cancer Res. (1997), 57(12), 2446-2451
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The mitochondrial permeability transition and oxidative stress seem to be crit. alterations in cellular physiol. that take place during programmed cell death. Failure to undergo apoptosis is assocd. with **drug** resistance in acute myeloid leukemia and other cancers. Therefore, it is important to establish causal relationships between the physiol. changes that take place in apoptosis, because these are potential targets for novel treatment strategies to overcome this form of **drug** resistance. We describe the use of multilaser flow cytometry methods to make correlated measurements of mitochondrial membrane potential (MMP), the generation of reactive oxygen intermediates, the cellular content of reduced glutathione (GSH), intracellular calcium, and exposure of phosphatidylserine on the cell surface. Using these combined methods, we have mapped a "death sequence" that occurs after treatment of leukemic blasts with clin. relevant concns. of 1-.beta.-D-arabinofuranosylcytosine (ara-C). Dual labeling of MMP and cellular glutathione content showed that loss of MMP, indicative of the permeability transition, took place in cells that were depleted of glutathione. The loss of MMP coincided with phosphatidylserine exposure and preceded a state of high reactive oxygen generation. Finally, there was an increase in intracellular calcium. These results demonstrate that the mitochondrial permeability transition takes place during ara-C toxicity but suggest that this occurs downstream of the loss of GSH. Thus, oxidative stress after ara-C-induced toxicity seems to be a biphasic phenomenon, with the permeability transition occurring after a depletion of GSH and preceding a state of high reactive oxygen generation.

IT **147-94-4**, Ara-c
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(relationships between **mitochondrial** permeability transition and oxidative stress during ara-C toxicity)

L7 ANSWER 22 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:39627 HCAPLUS
DOCUMENT NUMBER: 126:139552
TITLE: Bcl-2 and Bcl-XL antagonize the mitochondrial dysfunction preceding nuclear apoptosis induced by **chemotherapeutic** agents
AUTHOR(S): Decaudin, Didier; Geley, Stephan; Hirsch, Tamara;

Castedo, Maria; Marchetti, Philippe; Macho, Antonio; Kofler, Reinhard; Kroemer, Guido
CORPORATE SOURCE: Centre National de la Recherche Scientifique, Unite Propre de Recherche 420, Villejuif, F-94801, Fr.
SOURCE: Cancer Res. (1997), 57(1), 62-67
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A no. of apoptosis-inducing agents used in cancer **therapy** (etoposide, doxorubicin, 1-.beta.-D-arabinofuranosylcytosine), as well as the pro-apoptotic second messenger ceramide, induce a disruption of the mitochondrial transmembrane potential (.DELTA..psi.m) that precedes nuclear DNA fragmentation. This effect has been obsd. in tumor cell lines of T-lymphoid, B-lymphoid, and myelomonocytic origin in vitro. Circulating tumor cells from patients receiving **chemotherapy** in vivo also demonstrate a .DELTA..psi.m disruption after in vitro culture that precedes nuclear apoptosis. Transfection-enforced hyperexpression of the proto-oncogenes bcl-2 and bcl-XL protects against **chemotherapy** -induced apoptosis, at both the level of the mitochondrial dysfunction preceding nuclear apoptosis and the level of late nuclear apoptotic events. Bcl-2-mediated inhibition of ceramide-induced .DELTA..psi.m disruption is obsd. in normal as well as anucleate cells, indicating that bcl-2 acts on an extranuclear pathway of apoptosis. In contrast to Bcl-2 and Bcl-XL, hyperexpression of the protease inhibitor cytokine response modifier A fails to protect tumor cells against **chemotherapy** -induced .DELTA..psi.m disruption and apoptosis, although cytokine response modifier A does prevent the .DELTA..psi.m collapse and posterior nuclear apoptosis triggered by crosslinking of Fas/Apo-1/CD95. In conclusion, .DELTA..psi.m disruption seems to be an obligatory step of early (pre-nuclear) apoptosis, and .DELTA..psi.m is stabilized by two members of the bcl-2 gene family conferring resistance to **chemotherapy**.
IT **147-94-4**, 1-.beta.-D-Arabinofuranosylcytosine
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(bcl-2 and bcl-XL antagonize **mitochondrial** dysfunction preceding nuclear apoptosis induced by **chemotherapeutic** agents in human cells)

L7 ANSWER 23 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:652941 HCAPLUS
DOCUMENT NUMBER: 121:252941
TITLE: Morphological and functional analysis of rat cerebella with **drug**-induced deficit of Purkinje cells and granule cells during the developmental stages
AUTHOR(S): Takahashi, Megumi
CORPORATE SOURCE: School of Medicine, Yokohama City University, Yokohama, 236, Japan
SOURCE: Yokohama Med. Bull. (1993), 44(1-2), 57-72
CODEN: YMBUA7; ISSN: 0044-0531
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To investigate the cellular mechanism of ataxic symptom formation, the

studies of calcium imaging anal., immunostaining with anti-IP3 receptor (R) antibody (Ab), and immediate early genes (IEG) anal. were performed in two types of ataxic rats. One is Purkinje cell-deficit rats treated with methylazoxymethanol (MAM) and the other is granule cell-deficit rats treated with cytosine arabinoside (Ara C). Both of MAM-and Ara C-treated rats showed malnutrition and decrease of mobility. Ara C-treated rats showed more severe cerebellar symptoms, such as tremor and loss of cooperative motion. Calcium-imaging anal. demonstrated that the responses for NMDA and quisqualate (QA) in MAM-treated rat cerebella were almost similar to those in untreated rat cerebella, but that they were lost in Ara C-treated rat cerebella on postnatal day (PND) 21. Anti-IP3r Ab staining revealed that some Purkinje cells were found even in the internal granular layer on PND 14 and PND 21 in MAM-treated rats, and that Purkinje dendrites extended in random directions on PND 14 and were destroyed on PND 21 in Ara C-treated rats. IEG anal. (four IEG; c-myc, c-fos, c-jun and jun B) in MAM-treated rats showed minor changes in c-fos and c-jun mRNA expression patterns. In Ara C-treated rats c-fos mRNA level increased transiently on PND 18, and c-jun and jun-B mRNA levels were constantly low. Apparently, the degree of functional disorders in the rat cerebellum is well correlated to the severity of cerebellar symptoms, and IEG anal. is a most sensitive detection method of these functional disorders.

IT **147-94-4**, Cytosine arabinoside
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(cerebellar function in cytosine arabinoside- and methylazoxymethanol-induced **ataxia** in rats)

L7 ANSWER 24 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:645335 HCAPLUS
DOCUMENT NUMBER: 121:245335
TITLE: Anti-mitochondrial effects of bisethyl polyamines in mammalian cells
AUTHOR(S): Snyder, Ronald D.; Beach, Dorothy C.; Loudy, David E.
CORPORATE SOURCE: Marion Merrell Dow Res. Inst., Cincinnati, OH, 45215, USA
SOURCE: Anticancer Res. (1994), 14(2A), 347-56
CODEN: ANTRD4; ISSN: 0250-7005
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of three bisethyl polyamine analogs on mitochondrial structure and function were examd. in human HeLa and L1210 murine leukemia cells. N, N'-Bis-[3(ethylamino)-propyl]1-7-heptane diamine (BEPH), and its octane (BEPO), and butane (BESPM) deriv., were shown by electron microscopy and/or rhodamine 123 uptake studies to alter the structural integrity of mitochondria when both cell lines were treated at the approx. Ic50 dose of each **drug**. At this dose, BEPH had no marked effects on levels of the naturally occurring polyamines, putrescine, spermidine or spermine, in either cell line whereas BEPO and BESPM treatment did result in pool depletion. Southern blot anal. demonstrated a time and dose-dependent loss of mitochondrial DNA from BEPH-treated L1210 cultures suggesting that loss of mitochondrial integrity extended to the DNA level. Treatment of L1210 cells with all three analogs revealed marked redns. in the activity of two mitochondrial enzymes citrate synthase and cytochrome c oxidase.

HeLa cells treated with all three analogs exhibited markedly reduced levels of ATP, complete loss of cytidine triphosphate (CTP) and near total depletion of uridine triphosphate (UTP). There was also a loss of colony forming ability in HeLa cells which could be nearly completely reversed by the addn. of either uridine or cytidine suggesting that NTP redn. may be the primary antiproliferative determinant in these cells. Growth inhibition by BEPH In L1210 cells was markedly potentiated by the glycolysis inhibitor, 2-deoxyglucose, which had no such effect in otherwise untreated cells. This suggests that BEPH treatment of L1210 cells results in impairment of mitochondrial ATP synthesis and activation of the glycolytic pathway for energy prodn. 2-Deoxyglucose treatment also completely prevented the increase of ATP by BEPH treatment of L1210 cells. It is concluded that all three bisethyl polyamines alter HeLa and L1210 mitochondria both structurally and functionally and that these alterations may play a primary role in the antiproliferative activity of these agents in HeLa cells. In L1210, the different spectra of cellular biochem. changes following bisethyl polyamine treatment suggests that addnl. mechanisms may be in effect.

IT **63-39-8**, Uridine triphosphate **65-47-4**, Cytidine triphosphate
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(anti-**mitochondrial** effects of bisethyl polyamines for antineoplastics)

L7 ANSWER 25 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:598673 HCAPLUS
DOCUMENT NUMBER: 121:198673
TITLE: ATP-induced unspecific channel in yeast mitochondria
AUTHOR(S): Guerin, Bernard; Bunoust, Odile; Rouqueys, Valerie; Rigoulet, Michel
CORPORATE SOURCE: Inst. Biochim. Genet. Cell., Univ. Bordeaux 2, Bordeaux, 33077, Fr.
SOURCE: J. Biol. Chem. (1994), 269(41), 25406-10
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB ATP induced swelling of isolated yeast mitochondria suspended in an isoosmotic soln. of potassium gluconate. Valinomycin stimulated the swelling rate, indicating that K+ influx in the presence of ATP is rate-controlling. This swelling was inhibited by ADP, phosphate (probably acting on the external face of the inner membrane), and Mg2+, which forms a complex with ATP. ATP-induced swelling did not require working F0-F1-ATPase since it was not inhibited by oligomycin and uncoupler. CTP and GTP also induced a swelling. ATP also induced mitochondrial swelling in potassium glutamate, chloride, and acetate but not in phosphate solns. Sodium, but not ammonium, can replace potassium ion. It is probable that the ATP-channel opening also necessitates an electrogenic cation influx. Respiration also induced swelling of mitochondria suspended in isoosmotic potassium gluconate soln. ATP- or respiration-induced swelling were inhibited equally by N,N'-dicyclohexylcarbodiimide, propranolol, and Zn2+ but not by quinine; all these **drugs** inhibit the H+/K+ exchange. It was concluded that this unspecific channel is not open under conditions used to measure oxidative phosphorylation. Its physiol. role remains unknown.

IT **65-47-4**, 5'-CTP
RL: BIOL (Biological study)
(unspecific ion channel induction by, in yeast **mitochondria**)

L7 ANSWER 26 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:595680 HCAPLUS
DOCUMENT NUMBER: 121:195680
TITLE: Resistance to 1-.beta.-D-arabinofuranosylcytosine and hypersensitivity to bleomycin in ataxia telangiectasia B-lymphoblastoid cell lines
AUTHOR(S): Li, Ming Jie; Shiraishi, Yukimasa
CORPORATE SOURCE: Dep. Anat., Kochi Med. Sch., Nankoku, 783, Japan
SOURCE: Int. J. Oncol. (1994), 4(6), 1173-81
CODEN: IJONES; ISSN: 1019-6439
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Three ataxia telangiectasia (AT) B-lymphoblastoid cell lines (B-LCLs) were examd. for the chromosome aberrations induced by a DNA replication and repair inhibitor, 1-.beta.-D-arabinofuranosylcytosine (ara-C) and for the effects of ara-C on the frequencies of chromosome aberrations caused by bleomycin (BLM). All these AT cell lines exhibited resistance to ara-C compared with normal and Bloom syndrome (BS) cells. In contrast with the case in normal and BS cells, ara-C did not enhance chromosome aberrations produced by BLM in AT cells, although these cells showed hypersensitivity to BLM. After treatment with 1 x 10-5M ara-C for 24 h, total frequencies of chromosome aberrations in AT cells were 0.095-0.115/cell, which is about 6 times lower than those in normal (0.625/cell) and BS cells (0.775/cell). Following combination treatment with tetrahydrouridine (THU) and ara-C, the frequencies of chromosome aberrations in AT B-LCLs were greatly increased compared with those after treatment with ara-C alone. Furthermore, when AT cells were pretreated with THU in combination with ara-C, and then treated with BLM, a great synergistic enhancement of chromosome aberrations was obsd. Because THU is an exclusive inhibitor of cytidine deaminase, these results strongly indicate that in AT B-LCLs there could be overprodn. of cytidine deaminase, which is responsible for ara-C resistance. On the other hand, combination of THU and deoxycytidine (dCyd) reduced chromosome aberrations induced by BLM in AT cells, although dCyd alone had no effect on bleomycin-induced chromosome aberrations. Break point distributions on chromosome bands following treatment with BLM or ara-C plus THU, alone or in combination, were examd. and are discussed.
IT **147-94-4**, Ara-C **18771-50-1**, Tetrahydrouridine
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(resistance to ara-C and hypersensitivity to bleomycin in human **ataxia** telangiectasia lymphoblastoid cells)

L7 ANSWER 27 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:45253 HCAPLUS
DOCUMENT NUMBER: 120:45253
TITLE: Anti-human immunodeficiency virus type 1 **therapy** and peripheral neuropathy: Prevention of 2',3'-dideoxycytidine toxicity in PC12 cells, a neuronal model, by uridine and pyruvate
AUTHOR(S): Keilbaugh, Sue A.; Hobbs, Gregory A.; Simpson, Melvin

V.
CORPORATE SOURCE: Dep. Biochem. Cell Biol., State Univ. New York, Stony Brook, NY, 11794-5215, USA
SOURCE: Mol. Pharmacol. (1993), 44(4), 702-6
CODEN: MOPMA3; ISSN: 0026-895X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A strategy for preventing or delaying the peripheral neuropathy induced by 2',3'-dideoxycytidine (ddC) **therapy** in patients with acquired immunodeficiency syndrome was suggested by findings, in two labs., that cultured avian and mammalian cells devoid of mitochondrial DNA continue to replicate at virtually normal rates, provided that the medium is supplemented with uridine and pyruvate. Inasmuch as it is likely that a depletion of mitochondrial DNA also takes place in neuronal cells exposed to ddC, the authors used PC12 cells, the neuronal model the authors have reported on previously, in an attempt to rescue these cells from the deleterious effects of ddC. The authors first show, using undifferentiated PC12 cells, that DNA replication is impaired in mitochondria isolated from cells grown in the presence of ddC. Then, using growth rate as a criterion of the well-being of the cells, the authors show that the addn. of uridine and pyruvate to uninduced cells growing in the presence of ddC results in an av. rescue efficiency of 51%, based on the uridine/pyruvate-treated control. This value increases considerably at substantially higher concns. of uridine alone. Rescue efficiencies of differentiated cells, which do not proliferate, were assessed using neurite outgrowth and neurite survival as criteria. Here the rescue efficiency is 56%, based on the uridine/pyruvate-treated control. In addn., uridine and pyruvate prolong the viability of ddC-treated cells and maintain their healthy appearance; without these compds., the ddC-treated cells have an abnormal morphol. and die off quite rapidly.

IT **58-96-8**, Uridine
RL: BIOL (Biological study)
(prevention of dideoxycytidine DNA replication inhibition in PC12 cells, prevention of **neuropathy** in relation to)

L7 ANSWER 28 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1994:26108 HCAPLUS
DOCUMENT NUMBER: 120:26108
TITLE: Purification and characterization of deoxycytidine kinase from acute myeloid leukemia cell mitochondria
AUTHOR(S): Wang, Li-Ming; Kucera, Gregory L.; Capizzi, Robert L.
CORPORATE SOURCE: Comprehensive Cancer Center of Wake Forest University, Bowman Gray School of Medicine, Winston-Salem, NC, USA
SOURCE: Biochim. Biophys. Acta (1993), 1202(2), 309-16
CODEN: BBACAQ; ISSN: 0006-3002
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Deoxycytidine kinase is a key anabolic enzyme for the activation of ara-C (1-.beta.-D-arabinofuranosylcytosine) and other antitumor **drugs**, as well as normal purine and pyrimidine deoxynucleosides. Previously, two forms of the kinase have been identified; deoxycytidine kinase I (70 kDa) and deoxycytidine kinase II (70 kDa). Deoxycytidine kinase I utilized dCyd and ara-C as substrates, while deoxycytidine kinase II used dCyd and

dThd as substrates. Deoxycytidine kinase kinase II had very low activity on ara-C as a substrate. The authors report a procedure for the purifn. of a novel deoxycytidine kinase (52 kDa) from isolated human peripheral blood leukemia cell mitochondria. This enzyme has activity similar to deoxycytidine kinase II. The enzyme was extd. from the mitochondria with digitonin (1 mg/8 mg protein) and 0.3 M NaCl, and the ext. was purified by DEAE-cellulose chromatog. and thymidine-Sepharose affinity chromatog. This procedure produced a near homogeneous enzyme prepn. with a yield of 70%. The mitochondrial deoxycytidine kinase was localized to the outer mitochondrial membrane. The enzyme phosphorylated dCyd (Km = 17 .mu.M), however, ara-C was not a good substrate for the mitochondrial deoxycytidine kinase. ATP was the best phosphate donor, whereas dCTP and dTTP were potent inhibitors of mitochondrial deoxycytidine kinase. In contrast, phosphorylation of ara-C by deoxycytidine kinase I utilized GTP, dGTP, or ATP as a phosphate donor.

IT **147-94-4**, 1-.beta.-D-Arabinofuranosylcytosine
RL: BIOL (Biological study)
(deoxycytidine kinase of **mitochondria** of acute myeloid leukemia cells of human in relation to)

L7 ANSWER 29 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:34821 HCAPLUS
DOCUMENT NUMBER: 118:34821
TITLE: Selective assay for thymidine kinase 1 and 2 and deoxycytidine kinase and their activities in extracts from human cells and tissues
AUTHOR(S): Arner, Elias S. J.; Spasokukotskaya, T.; Eriksson, Staffan
CORPORATE SOURCE: Med. Nobel Inst., Karolinska Inst., Stockholm, S-104 01, Swed.
SOURCE: Biochem. Biophys. Res. Commun. (1992), 188(2), 712-18
CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Human cells salvage pyrimidine deoxyribonucleosides via 5'-phosphorylation which is also the route of activation of many **chemotherapeutically** used nucleoside analogs. Key enzymes in this metab. are the cytosolic thymidine kinase (TK1), the mitochondrial thymidine kinase (TK2) and the cytosolic deoxycytidine kinase (dCK). These enzymes are expressed differently in different tissues and cell cycle phases, and they display overlapping substrate specificities. Thymidine is phosphorylated by both thymidine kinases, and deoxycytidine is phosphorylated by both dCK and TK2. The enzymes also phosphorylate nucleoside analogs with very different efficiencies. Here the authors present specific radiochem. assays for the 3 kinase activities utilizing analogs as substrates that are by more than 90% phosphorylated solely by one of the kinases; i.e. e'-azido-2',3'-dideoxythymidine (AZT) as substrate for TK1, 1-.beta.-D-arabinofuranosylthymidine (AraT) for TK2 and 2-chlorodeoxyadenosine (CdA) for dCK. The fraction of the total deoxycytidine and thymidine phosphorylating activity that was provided by each of the 3 enzymes in different human cells and tissues, such as resting and proliferating lymphocytes, lymphocytic cells of leukemia patients (chronic lymphocytic, chronic myeloic and hairy cell leukemia), muscle brain and gastrointestinal tissue was detd. The detailed knowledge

of the pyrimidine deoxyribonucleoside kinase activities and substrate specificities are of importance for studies on **chemotherapeutically** active nucleoside analogs, and the assays and data presented here should be valuable tools in that research.

IT **605-23-2**
RL: ANST (Analytical study)
(in thymidine kinase **mitochondrial** isoenzyme radiochem. detn., as substrate)

L7 ANSWER 30 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:464089 HCAPLUS
DOCUMENT NUMBER: 115:64089
TITLE: Effect of anti-human immunodeficiency virus nucleoside analogs on mitochondrial DNA and its implication for delayed toxicity
AUTHOR(S): Chen, Chin Ho; Vazquez-Padua, Miguel; Cheng, Yung Chi
CORPORATE SOURCE: Sch. Med., Yale Univ., New Haven, CT, 06510, USA
SOURCE: Mol. Pharmacol. (1991), 39(5), 625-8
CODEN: MOPMA3; ISSN: 0026-895X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The anti-human immunodeficiency virus (anti-HIV) nucleoside analogs azidothymidien (AZT), dideoxycytidine (ddC), dideoxyinosine (ddI), dideoxydidehydrothymidine (D4T), and dideoxydidehydrocytidine (D4C) and the anticancer **drug** cytosine arabinoside (AraC) were compared for their effects on the mitochondrial DNA (mtDNA) content in a human lymphoblastoid cell line, CEM. The potency of these compds. in reducing mtDNA content was in the order of ddC > D4C > D4T > AZT > ddI. AraC did not have a significant effect on mtDNA content. All of the compds. tested, except AraC, stimulated lactic acid prodn. at concns. that inhibited mtDNA synthesis. The action of ddC and ddI occurred at concns. that did not affect cell growth significantly in 4 days but retarded cell growth by day 6. D4T and D4C decreased mtDNA content by 50% at doses lower than those that inhibited cell growth by 50% in 4 days (ID50). However, AZT required a dose higher than the ID50 to exert similar effects on mtDNA content. The decrease of mtDNA content caused by ddC also occurred in nerve growth factor-treated PC12 cells, which differentiate to neuron-like cells upon treatment with nerve growth factor. The preferential inhibition of mtDNA, compared with cell growth, by some of these anti-HIV nucleoside analogs correlates well with their ability to cause **drug**-limiting delayed toxicity, such as peripheral neuropathy, in patients. These data suggest that the selective mitochondrial toxicity could be responsible for the delayed toxicity caused by these anti-HIV analogs.

IT **147-94-4**, AraC
RL: BIOL (Biological study)
(as anti-HIV agent, **mitochondrial** DNA decrease by, delayed toxicity in relation to)

L7 ANSWER 31 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:567671 HCAPLUS
DOCUMENT NUMBER: 111:167671
TITLE: Effect of YM-14673, a new thyrotropin-releasing hormone analog, on ataxic gait in cytosine

arabinoside-treated mice

AUTHOR(S): Yamamoto, Minoru; Shimizu, Masao

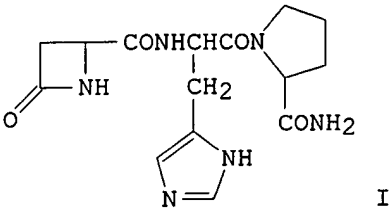
CORPORATE SOURCE: Dep. Pharmacol., Yamanouchi Pharm. Co. Ltd., Ibaraki, Japan

SOURCE: Eur. J. Pharmacol. (1989), 166(3), 545-7
CODEN: EJPHAZ; ISSN: 0014-2999

DOCUMENT TYPE: Journal

LANGUAGE: English

GI



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AB The effect of YM-14673 (I), a new TSH-releasing hormone (TRH) analog, on ataxic gait in mice treated with cytosine arabinoside was compared with the effect of TRH. Ataxic gait was obsd. after administration of cytosine arabinoside in a dose of 40 mg/kg (s.c.) on the 2nd and 3rd postnatal days. The falling index, the ratio of the no. of inversions to spontaneous motor activity, is regarded as an index of ataxia. TRH or YM-14673 administered i.p. 4-5 wk after the cytosine arabinoside reduced the falling index, with YM-14673 being about 30 times more potent than TRH in reducing the ataxic activity. Thus, YM-14673 ameliorates the ataxic gait of cytosine arabinoside-treated mice, suggesting that it may be of **therapeutic** use for treatment of patients with spino-cerebellar degeneration.

IT **147-94-4**, Cytosine arabinoside
RL: BIOL (Biological study)
(**ataxia** from, TRH and TRH analog effect on)

L7 ANSWER 32 OF 48 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1988:215980 HCAPLUS

DOCUMENT NUMBER: 108:215980

TITLE: 'Petite' mutagenesis by anticancer **drugs** in Saccharomyces cerevisiae

AUTHOR(S): Ferguson, Lynnette R.; Turner, Pamela M.

CORPORATE SOURCE: Med. Sch., Univ. Auckland, Auckland, N. Z.

SOURCE: Eur. J. Cancer Clin. Oncol. (1988), 24(4), 591-6
CODEN: EJCODS; ISSN: 0277-5379

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The antimitochondrial effects of a range of current clin. and exptl. antitumor **drugs** with varying modes of action were tested by using the petite mutagenesis model in S. cerevisiae. Of agents currently in the clinic, the antimetabolites 5-fluorouracil and methotrexate were extremely effective in inducing this respiratory defect, providing cells were growing during treatment. Adriamycin, BCNU, bleomycin, methyl-CCNU,

cis-platinum, chlorambucil, daunomycin, nitracine, N mustard, and hycanthone were also weakly effective petite mutagens, in either growing or nongrowing conditions. None of the currently used agents but some exptl. **drugs** induced high nos. of petite mutants during growing or non-growing conditions. To date, where such agents have been tested clin., they have proved either ineffective or very toxic. It is possible that antimitochondrial effects on nonproliferating cellular tissues such as the heart might cause unacceptable toxicity and preclude the clin. use of such agents. For those agents effective against proliferating cells, the mitochondria could be an important target for **chemotherapy** in some cell types. This type of **drug** appears relatively uncommon in the clinic at present. The petite mutagenesis assay could be more widely used as a screen to optimize this property in development of analogs of current clin. agents, or in developing new types of anticancer **drug**.

IT **147-94-4**, Cytosine arabinoside
RL: PRP (Properties)
(**mitochondria** anti-DNA effects of)

L7 ANSWER 33 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:618580 HCAPLUS
DOCUMENT NUMBER: 105:218580
TITLE: Depression of cytochrome P-450 and alterations of protein metabolism in mice treated with the interferon inducer polyriboinosinic acid.cntdot.polyribocytidylic acid
AUTHOR(S): Gooderham, Nigel J.; Mannering, Gilbert J.
CORPORATE SOURCE: Med. Sch., Univ. Minnesota, Minneapolis, MN, 55455, USA
SOURCE: Arch. Biochem. Biophys. (1986), 250(2), 418-25
CODEN: ABBIA4; ISSN: 0003-9861
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Treatment of mice with the interferon inducer poly(IC) [**24939-03-5**] results in the depression of several hepatic proteins. In this study the authors examd. synthesis and degrdn. of the proteins of liver cell organelles in mice treated with poly(IC). Effects on synthesis were detd. by using [14C]- and L-[3H]leucine incorporation into control and poly(IC)-treated mice, resp. At selected times after poly(IC) treatment the 3H/14C ratio was established for prepns. of nuclei, **mitochondria**, lysosomes, smooth endoplasmic reticulum, rough endoplasmic reticulum, and 105,000g supernatant (cytosol). Time-dependent alterations in de novo protein synthesis were greatest in lysosomal and rough endoplasmic reticular fractions; both were depressed 9 h after treatment. The effects of poly(IC) on protein degrdn. were detd. with [14C]bicarbonate. Poly(IC) treatment decreased the time required for disappeared of 50% of 14C-labeled protein of smooth and rough endoplasmic reticula. Examn. of endoplasmic reticulum marker enzymes showed depression of cytochrome P 450 [9035-51-2] and cytochrome b5 [9035-39-6] from 9 h onward after poly(IC) administration. Tyrosine aminotransferase [9014-55-5] activity was elevated 6 h after treatment with poly(IC), and then depressed after 9 h. The other organelle marker enzymes were not affected. Thus, poly(IC) decreases the content of proteins of the hepatic endoplasmic reticulum, including certain cytochrome P 450 isozymes, by

decreasing rates of protein synthesis and increasing rates of protein degrdn.

L7 ANSWER 34 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:417985 HCAPLUS
DOCUMENT NUMBER: 105:17985
TITLE: The effect of cytosine arabinoside upon mitochondrial staining kinetics in human hematopoietic cells
AUTHOR(S): Haanen, C.; Muus, P.; Pennings, A.
CORPORATE SOURCE: Dep. Intern. Med., Univ. Hosp. St. Radboud, Nijmegen, NL-6500 HB, Neth.
SOURCE: Histochemistry (1986), 84(4-6), 609-13
CODEN: HCMYAL; ISSN: 0301-5564
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The measurement of time-correlated intracellular **mitochondrial** staining with 3,3'-dipentyloxacarbocyanine [Di-O-C5(3)] appeared of interest to defne the optimal staining conditions. **Mitochondrial** staining of lymphocytes, monocytes, and granulocytes results in different fluorescence signals, related to the nos. of **mitochondria**, that are present in the cells of these various cell types. Alterations of Di-O-C(5)3 staining in a distinct cell type are due to changes in the physiol. or functional state of the **mitochondria**. It appeared that such alterations occur in cells, which are cultured in the presence of cytosine arabinoside [**147-94-4**]. The effect of cytotoxic **drugs** upon the **mitochondrial** membrane potential may be of relevance for understanding the mechanism of the action exerted by cytotoxic **drugs** upon cell biol.
IT **147-94-4**
RL: BIOL (Biological study)
(**mitochondrial** membrane potential in human hematopoietic cells response to)

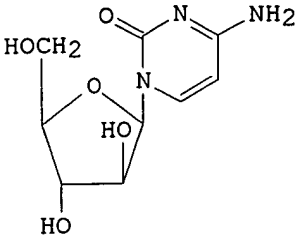
L7 ANSWER 35 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:45278 HCAPLUS
DOCUMENT NUMBER: 104:45278
TITLE: Transport and metabolism of double-labeled CDP-choline in mammalian tissues
AUTHOR(S): Galletti, Patrizia; De Rosa, Mario; Ausilia Nappi, Maria; Pontoni, Gabriele; Del Piano, Luisa; Salluzzo, Antonio; Zappia, Vincenzo
CORPORATE SOURCE: 1st Med. Sch., Univ. Naples, Naples, 80138, Italy
SOURCE: Biochem. Pharmacol. (1985), 34(23), 4121-30
CODEN: BCPCA6; ISSN: 0006-2952
DOCUMENT TYPE: Journal
LANGUAGE: English
AB [Methyl-14C,5-3H]CDP-choline [**99874-02-9**] was synthesized and subjected to a **pharmacokinetic** anal. in several biol. systems. In transport expts. with intact human erythrocytes no incorporation of radioactivity is observable. On the other hand the results obtained with perfused rat liver suggest a rapid cleavage of the pyrophosphate bridge of the mol., followed by a rapid uptake of the hydrolytic products. The plasma half-lives of i.v. injected CDP-choline and of its metabolites were evaluated within 60-s range. Renal and fecal excretion of the injected

radioactivity is negligible: only 2.5% of the administered 14C and 6.5% of the 3H is excreted up to 48 h after administration. Liver and kidney are the major CDP-choline-metabolizing organs, characterized by a fast and extensive uptake of choline metabolites, followed by a slow release; conversely the rate of uptake of both the 3H- and 14C-labeled moieties by rat brain is significantly slower, reaching a steady-state level after 10 h. The characterization of the labeled compds. detectable in the investigated organs provides some insights into the metab. of the **drug**: (i) the 3H-cytidine moiety in all the examd. organs appears to be incorporated into the nucleic acid fraction via the cytidine nucleotide pool; (ii) the [14C]choline moiety of the mol. is in part converted, at the **mitochondrial** level, into betaine [107-43-7], which accounts for about 60% of the total 14C-radioactivity assocd. with liver and kidney 30 min after administration; (iii) [14C]betaine in turn acts as Me donor to homocysteine, yielding methionine [63-68-3], subsequently incorporated into proteins; (i.v.) the time-dependent increase in labeled phospholipids is indicative of a recycling of the choline Me groups in this lipid fraction via CDP-choline and/or S-adenosylmethionine; (v) the rather extensive amt. of labeled methionine detectable in brain probably arises from its uptake from the blood, since the enzyme catalyzing the conversion of betaine to methionine is lacking in brain.

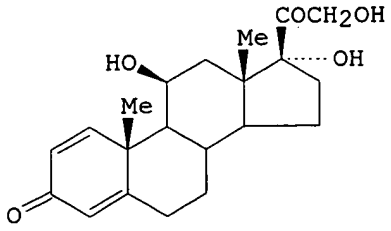
L7 ANSWER 36 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1985:142887 HCAPLUS
DOCUMENT NUMBER: 102:142887
TITLE: Studies with the IFN inducer and immune modulator, poly ICLC
AUTHOR(S): Levy, H. B.; Chirigos, M.
CORPORATE SOURCE: NIAID, Frederick, MD, USA
SOURCE: Contrib. Oncol. (1984), 20(Physiol. Pathol. Interferon Syst.), 358-74
CODEN: COONEV
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The **pharmacol**. of poly ICLC (polyI.cntdot. poly C complex with poly lysine and CM-cellulose) [**64685-78-5**] in humans and lab. animals is described. Poly ICLC has **therapeutic** value in viral diseases in mice and monkeys and causes modification of a no. of immune parameters, both humoral and cell assocd. It also induces interferon formation in humans. The use of poly ICLC for treatment of cancer and several neurol. diseases whose etiol. is presumed to be assocd. with immune dystrophy (peripheral paralytic **neuropathy** and muscular dystrophy) is described.

L7 ANSWER 37 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:569167 HCAPLUS
DOCUMENT NUMBER: 99:169167
TITLE: Respiratory function of liver mitochondria in experimental leukemia. Effect of ascites of L1210 leukemic mice and antileukemic agents on isolated liver mitochondria
AUTHOR(S): Takatsuki, Yoshio
CORPORATE SOURCE: Sch. Med., Toho Univ., Tokyo, Japan

SOURCE: Toho Igakkai Zasshi (1983), 30(2), 197-209
CODEN: TOIZAG; ISSN: 0040-8670
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
GI



II



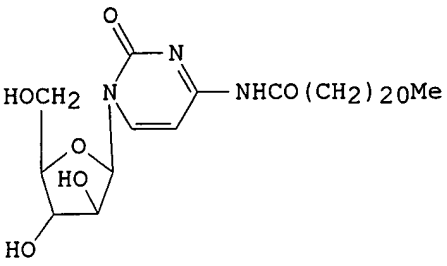
III

AB The influence of ascites obtained from L1210 leukemic mice on both the respiratory function and electron microscopic features of the liver **mitochondria** isolated from healthy DONRYU rats was studied. In addn., the influence of antileukemic agents on the liver **mitochondria** of BDF1 mice was examd. The introduction of nontreated ascites depressed the **mitochondrial** respiratory function causing an uncoupling phenomenon, whereas the addn. of urea-treated ascites markedly enhanced the **mitochondrial** respiratory function. Large amts. of inosine (I) [58-63-9] were isolated from the ascites. This suggests that the uncoupling phenomenon obsd. after the addn. of ascites might be induced by I acting as an uncoupling agent which activates silent ATPase. When compared with control under electron microscopy, the nontreated ascites-added **mitochondria** had an irregular shape, wider spaces between the inner and outer layers of the membrane, a loss of or shortened cristae, and a decreased electron d. of the matrix. The respiratory function of the liver **mitochondria** , after the addn. of i.v. injection of antileukemic agents, was significantly lowered in the Ara-C (II) [**147-94-4**] group when compared with the control group, but was markedly enhanced in the group given II plus prednisolone (III) [50-24-8].

IT **147-94-4**
RL: BIOL (Biological study)
(respiration by liver **mitochondria** response to prdnisolone and, antileukemic activity in relation to)

L7 ANSWER 38 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:463818 HCAPLUS
DOCUMENT NUMBER: 99:63818
TITLE: Intracellular distribution of N4-behenoyl-1-.beta.-D-arabinofuranosylcytosine in blood cells
AUTHOR(S): Ueda, Takanori; Nakamura, Toru; Kagawa, Daizaburo; Yamamoto, Kokichi; Uchida, Michihiko; Sasada, Masataka; Uchino, Haruto
CORPORATE SOURCE: Fac. Med., Kyoto Univ., Kyoto, 606, Japan
SOURCE: Gann (1983), 74(3), 445-51
CODEN: GANNA2; ISSN: 0016-450X

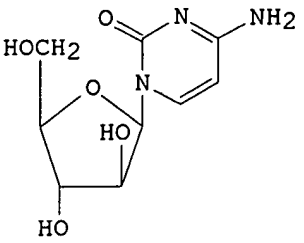
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB In order to clarify differences in the mode of action between N4-behenoyl-1-.beta.-D-arabinofuranosylcytosine (behenoyl-ara-C) (I) [55726-47-1] and 1-.beta.-D-arabinofuranosylcytosine (ara-C) [**147-94-4**], comparative studies on both agents were undertaken. When human erythrocytes incubated with behenoyl-ara-C-acyl-1-14C were fractionated into stroma and stroma-free lysate, a marked accumulation of radioactivity in stroma was obsd. In contrast, ara-C-cytosine-2-14C was rapidly incorporated into the stroma-free lysate. This-layer chromatog. of the exts. of leukemia L1210 cells incubated with behenoyl-ara-C-acyl-1-14C or behenoyl-ara-C-cytosine-2-14C at 37.degree. for 60 min revealed that most of the incorporated **drug** remained as unmetabolized behenoyl-ara-C. After incubation of 20 .mu.M behenoyl-ara-C or ara-C with L1210 cells at 37.degree. for 60 min, subcellular fractionation of the cell suspension was performed; behenoyl-ara-C was accumulated markedly in the membrane, **mitochondria**, and microsome fractions. In contrast, most of the ara-C was found in the 105,000 g supernatant fraction. The accumulation of behenoyl-ara-C in membrane structures may result from the lipophilic nature of the agent, which may have a prolonged inhibitory action on leukemic cell proliferation.

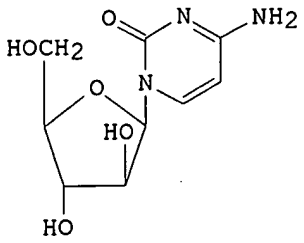
L7 ANSWER 39 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:447615 HCAPLUS
DOCUMENT NUMBER: 99:47615
TITLE: In vivo effects of cytosine arabinoside on deoxyribonucleic acid replication in Chinese hamster ovary cells. 2. Cytosine arabinoside affects the rate of synthesis but not the pattern of labeling of an amplified chromosomal sequence at the onset of the S period
AUTHOR(S): Heintz, Nicholas H.; Hamlin, Joyce L.
CORPORATE SOURCE: Sch. Med., Univ. Virginia, Charlottesville, VA, 22908, USA
SOURCE: Biochemistry (1983), 22(15), 3557-62
CODEN: BICHAW; ISSN: 0006-2960
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB The effect ara-C (I) [**147-94-4**] on DNA replication in methotrexate-resistant Chinese hamster ovary cells was examd. under circumstances in which nuclear DNA synthesis could be distinguished from **mitochondrial** DNA synthesis. G1-arrested cells were induced to traverse G1 and enter the S phase in the presence of radiolabeled thymidine and various concns. of the **drug**. Ara-C did not affect the kinetics of G1 traverse and subsequent entry into S after release from isoleucine deprivation, as measured by autoradiog. However, the inhibitor reduced the net rate of thymidine incorporation into nuclear DNA in a dose-dependent fashion. Autoradiog. of nuclear matrix-DNA halo structures suggests that the **drug** inhibits nuclear thymidine incorporation by slowing chain elongation and movement of newly replicated DNA through a matrix-bound replication app. Southern blot anal. of restriction digests of DNA radiolabeled in early S in the presence of ara-C indicates that the synthesis of the early-replicating amplified dihydrofolate reductase domain in these cells begins at sequences identical with those obsd. in cells synchronized with aphidicolin or hydroxyurea. Progressively lower concns. of ara-C permit proportionately greater extents of the amplified unit to be replicated. Apparently, ara-C slows the rate of chain elongation without altering the site at which DNA replication is initiated within individual replicons.

L7 ANSWER 40 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:447614 HCAPLUS
DOCUMENT NUMBER: 99:47614
TITLE: In vivo effects of cytosine arabinoside on deoxyribonucleic acid replication in Chinese hamster ovary cells. 1. Resolution of differential effects on mitochondrial and nuclear deoxyribonucleic acid synthesis
AUTHOR(S): Heintz, Nicholas H.; Hamlin, Joyce L.
CORPORATE SOURCE: Sch. Med., Univ. Virginia, Charlottesville, VA, 22908, USA
SOURCE: Biochemistry (1983), 22(15), 3552-7
CODEN: BICHAW; ISSN: 0006-2960
DOCUMENT TYPE: Journal
LANGUAGE: English
GI

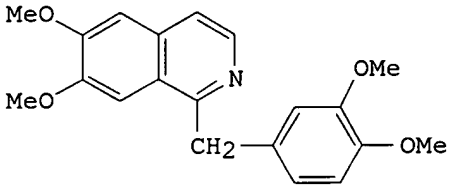


I

AB The effect of ara C (I) [**147-94-4**] on the uptake of radiolabeled thymidine in Chinese hamster ovary (CHO) cells entering the S period was examd. The inhibition of thymidine incorporation into DNA by ara-C had a biphasic dose-response curve. Characterization of DNA synthesized in the presence of the **drug** by alk. sucrose gradient sedimentation demonstrated a refractile component at concns. >1.0 .mu.g/mL. Restriction digestion of DNA, followed by electrophoresis, Southern transfer, and autoradiog., indicated that as the concn. of ara-C increases, thymidine incorporation is progressively limited to 3 EcoR1 fragments whose total length is approx. 15.8 kilobase pairs. Furthermore, DNA labeled with [3H]thymidine in a high concn. of ara-C was shown to band at a heavier position than main-band DNA in neutral CsCl gradients. Labeling of DNA in CHO cells that lack a functional nuclear thymidine kinase gene suggested that the component whose synthesis is insensitive to the inhibitory action of ara-C is **mitochondrial** in origin. This suggestion was confirmed by demonstrating that restriction fragments that are labeled in high concns. of ara-C hybridize to 32P-labeled Chinese hamster **mitochondrial** DNA (mtDNA). These results were obtained with nuclear DNA prepd. by std. methods and indicate that the study of the mode of action of ara-C on DNA synthesis in mammalian cells is complicated by the presence of mtDNA, whose synthesis is at least 50-fold less sensitive to the action of the inhibitor than is nuclear DNA replication.

IT **147-94-4**
RL: BIOL (Biological study)
(DNA formation by cell nucleus and **mitochondria** response to)

L7 ANSWER 41 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:400503 HCAPLUS
DOCUMENT NUMBER: 99:503
TITLE: Differentiated **pharmacological** action as a function of age on cerebral enzymatic activities related to energy transduction
AUTHOR(S): Benzi, G.; Arrigoni, E.; Dagani, F.; Marzatico, F.; Curti, D.; Polgatti, M.; Villa, R. F.; Agnoli, A.
CORPORATE SOURCE: Ist. Farm., Fac. Sci., Pavia, 27100, Italy
SOURCE: Alpha-Bloquants, Symp. Int. (1981), Meeting Date 1979, 362-72. Masson: Paris, Fr.
CODEN: 49LNA7
DOCUMENT TYPE: Conference
LANGUAGE: English
GI



I

AB The effect of **drugs** on cerebral enzyme activities was studied in rats with respect to age (young adult to senescence). One-month treatment of rats with papaverine (I) [58-74-2] at 20 wk of age increased lactate dehydrogenase [9001-60-9], malate dehydrogenase [9001-64-3], and cytochrome oxidase [9001-16-5] in brain homogenate. At 60 and 100 wk, the same treatment led to enhancement of only lactate dehydrogenase and cytochrome oxidase; no effect was obsd. at 140 wk. One-month treatment with theophylline propanesulfonate [1672-28-2] affected only lactate dehydrogenase at 20, 60, and 100 wk of age; no effect was detected at 140 wk. One-month treatment with trimetazidine [5011-34-7] caused, at 20 wk, inhibition of all enzyme activities in the **mitochondrial** fraction and inhibition of cytochrome oxidase in the homogenate. Subsequently (60 and 100 wk of age) only cytochrome oxidase in the homogenate and citrate synthase [9027-96-7] were inhibited. No inhibition was obsd. at 140 wk of age. After 1-mo treatment with nicergoline tartrate [32222-75-6] at 20 wk, cytochrome oxidase, malate dehydrogenase, and citrate synthase were inhibited, while the activity of NADH-cytochrome c reductase [9027-14-9] appeared to be increased. At 60 and 100 wk of age, these effects were seen only with total NADH-cytochrome c reductase and citrate synthase, while at 140 wk they were no longer detected. One-month treatment with cytidine diphosphate choline [**987-78-0**] led to inhibition of **mitochondrial** citrate synthase and this was still evident at 140 wk. One-month treatment with vincamine theophylline propanesulfonate [51179-28-3] increased enzyme activity at 20, 60, and 100 wk of age. At 140 wk only lactate dehydrogenase and cytochrome oxidase were still elevated. Apparently age progressively narrows the range of **drug** effects on enzyme activities in brain. The classification of **drug** action on brain enzyme activities must necessarily take into account the age of the animal.

L7 ANSWER 42 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:574591 HCAPLUS
DOCUMENT NUMBER: 97:174591
TITLE: Monitoring the effect of anticancer **drugs** on L1210 cells by a mitochondrial probe, rhodamine-123
AUTHOR(S): Bernal, Samuel D.; Shapiro, Howard M.; Chen, Lan Bo
CORPORATE SOURCE: Sidney Farber Cancer Inst., Harvard Med. Sch., Boston, MA, 02115, USA
SOURCE: Int. J. Cancer (1982), 30(2), 219-24
CODEN: IJCNAW; ISSN: 0020-7136
DOCUMENT TYPE: Journal
LANGUAGE: English

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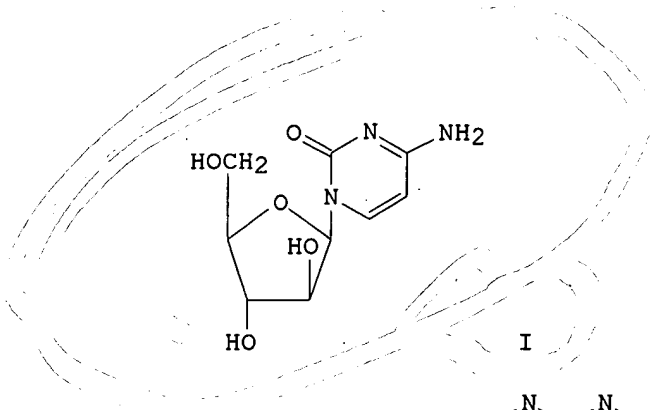

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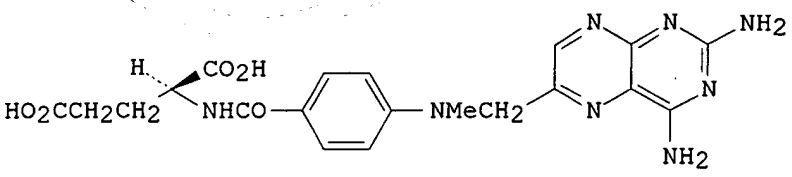

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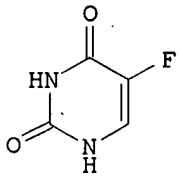


III

AB The cancer **chemotherapeutic** agents ara-C (I) [**147-94-4**], methotrexate (II) [59-05-2], and 5-FU (III) [51-21-8] cause a rapid loss of **mitochondrial** Rh-123 uptake in L1210 cells, which correlates with the loss of clonogenic ability. The loss of Rh-123 uptake is irreversible and occurs prior to Trypan Blue staining. Thus, the antimetabolites, unlike freeze-thawing and detergent treatments, generally cause **mitochondrial** damage prior to changes in plasma membrane permeability. Since the effect of antimetabolites on Rh-123 uptake is maximal at 24 h, the Rh-123 assay may provide a rapid alternative to the clonogenic assay for monitoring the cytotoxic effects of these **drugs**. The inhibition or impairment of **mitochondrial** function may be an important step in the cytocidal and(or) cytostatic action of anticancer **drugs**.

IT **147-94-4**
RL: PRP (Properties)
(cytotoxicity of, **mitochondrial** rhodamine-123 uptake in evaluation of)

L7 ANSWER 43 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:178644 HCAPLUS
DOCUMENT NUMBER: 96:178644
TITLE: On the contribution of the mitochondrial genome to the growth of Chinese hamster embryo cells in culture

AUTHOR(S): Morais, Rejean; Guertin, Denise; Kornblatt, Jack A.
CORPORATE SOURCE: Inst. Cancer Montreal, Hop. Notre-Dame, Montreal, PQ, H2L 4M1, Can.
SOURCE: Can. J. Biochem. (1982), 60(3), 290-4
CODEN: CJBIAE; ISSN: 0008-4018
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Chinese hamster embryo cell populations in culture can be adapted to grow in the presence of chloramphenicol. Tryptose phosphate broth and uridine, one of its components, prevent the growth-inhibitory effect of the **drug**. Study of some respiratory parameters (cytochrome c oxidase, cytochrome spectra, and O consumption) indicated that neither the broth nor uridine prevented the inhibitory effect of chloramphenicol on mitoribosomal protein synthesis. The cells grew with mitochondria devoid of a functional respiratory chain. Auxotrophy for pyrimidines appeared to result from the absence of dihydroorotate dehydrogenase, a respiratory chain-linked enzyme that catalyzes the 4th step of de novo pyrimidine biosynthesis. The synthesis of orotic acid may be considered as one of the main contributions of mitochondria to the growth of animal cells in culture.

IT **58-96-8**
RL: BIOL (Biological study)
(Chinese hamster embryo cell growth response to, in culture, **mitochondria** gene expression in relation to)

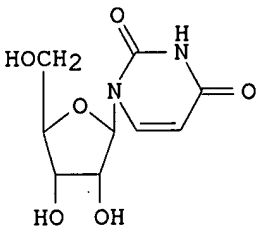
L7 ANSWER 44 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1981:167618 HCAPLUS
DOCUMENT NUMBER: 94:167618
TITLE: Aging and brain enzymes
AUTHOR(S): Benzi, G.; Arrigoni, E.; Dagani, F.; Marzatico, F.; Curti, D.; Polgatti, M.; Villa, R. F.
CORPORATE SOURCE: Inst. Pharmacol., Univ. Pavia, Pavia, Italy
SOURCE: Ettore Majorana Int. Sci. Ser.: Life Sci. (1980), 5(Aging Brain: Neurol. Ment. Disturbances), 1-13
CODEN: EMISDN; ISSN: 0199-9966
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Age-dependent changes of cerebral activities of lactate dehydrogenase [9001-60-9], citrate synthase [9027-96-7], and malate dehydrogenase [9001-64-3], NADH-cytochrome c reductase [9027-14-9] and cytochrome oxidase [9001-16-5] were studied in the homogenate and/or in the crude **mitochondrial** fraction of the brain of rats age 20, 60, 100 and 140 wk. With age, all the activities studied exhibited a decrease. Trimetazidine-2HCl [13171-25-0], papaverine-HCl [61-25-6], vincamine theophyllinylpropane sulfonate [51179-28-3], Na theophyllinylpropane sulfonate [77117-63-6], nicergoline tartrate [32222-75-6], and cytidine diphosphate choline [**987-78-0**] administered daily i.p. for 4 wk each (16-20, 56-60, 96-100 and 136-140 wk of life) at a dose level of 1 or 5 mg/kg exerted typical effects on the various enzymic activities of the brain. The range of **drug**-interference with these enzymic activities narrowed remarkably during maturity and even more during senescence.

L7 ANSWER 45 OF 48 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1980:597684 HCAPLUS
DOCUMENT NUMBER: 93:197684
TITLE: **Drug** interference on the age-dependent modification of the cerebral enzymic activities related to energy transduction
AUTHOR(S): Benzi, G.; Arrigoni, E.; Dagani, F.; Marzatico, F.; Curti, D.; Polgatti, M.; Villa, R. F.
CORPORATE SOURCE: Inst. Pharmacol., Univ. Pavia, Pavia, Italy
SOURCE: Aging (N. Y.) (1980), 13(Aging Brain Dementia), 113-17
CODEN: AGNYDE; ISSN: 0160-2721
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Age-dependent changes of some cerebral enzymic activities [lactate dehydrogenase (EC 1.1.1.27) [9001-60-9], citrate synthase (EC 4.1.3.7) [9027-96-7], malate dehydrogenase (EC 1.1.1.37) [9001-64-3], NADH-cytochrome c reductase (EC 1.6.99.3) [9079-67-8], and cytochrome oxidase (EC 1.9.3.1) [9001-16-5]] were studied in the whole homogenate and(or) in the crude **mitochondrial** fraction of the brain in rats aged 20, 60, 100, and 140 wk. With aging from youth to senescence, all the activities studied exhibited a natural decrease to low values. The **drugs** tested (papaverine-HCl [61-25-6], vincamine theophyllinylpropanesulfonate [75262-96-3], Na theophyllinylpropanesulfonate [75241-14-4], cytidine diphosphate choline [**987-78-0**], trimetazidine-2HCl [13171-25-0], and nicergoline tartrate [32222-75-6]) were administered daily for periods of 4 wk each (16-20, 56-60, 96-100, and 136-40 wk of life) i.p. and at 1 or 5 mg/kg. The **drugs** exerted specific effects on the various enzymic activities of the brain. The extent of **drug** interference with these enzymic activities decreased markedly during maturity and even more during senescence.

L7 ANSWER 46 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1980:488480 HCAPLUS
DOCUMENT NUMBER: 93:88480
TITLE: Chick embryo cells rendered respiration-deficient by chloramphenicol and ethidium bromide are auxotrophic for pyrimidines
AUTHOR(S): Morais, Rejean; Gregoire, Michel; Jeannotte, Lucie; Gravel, Denis
CORPORATE SOURCE: Inst. Cancer Montreal, Cent. Hosp. Notre-Dame, Montreal, PQ, H2L 4M1, Can.
SOURCE: Biochem. Biophys. Res. Commun. (1980), 94(1), 71-7
CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB Uridine (I) [**58-96-8**] confers on cultured chick embryo cells resistance to the growth inhibitory effect on chloramphenicol [56-75-7] and ethidium bromide [1239-45-8]. Cellular cytochrome oxidase [9001-16-5] activity is lost suggesting that uridine does not prevent the inhibitory effect of the **drugs** on **mitochondrial** transcription and translation. Other than cytidine [**65-46-3**], none of the precursors and derivs. of uridine tested supports cell growth.

L7 ANSWER 47 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1980:87939 HCAPLUS
DOCUMENT NUMBER: 92:87939
TITLE: Effect of chronic treatment with some **drugs** on the enzymatic activities of the rat brain
AUTHOR(S): Benzi, G.; Arrigoni, E.; Dagani, F.; Marzatico, F.; Curti, D.; Manzini, A.; Villa, R. F.
CORPORATE SOURCE: Inst. Pharmacol., Univ. Pavia, Pavia, Italy
SOURCE: Biochem. Pharmacol. (1979), 28(18), 2703-8
CODEN: BCPCA6; ISSN: 0006-2952
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Lactate dehydrogenase (EC 1.1.1.27) (I) [9001-60-9], citrate synthase (EC 4.1.3.7) (II) [9027-96-7], malate dehydrogenase (EC 1.1.1.37) (III) [9001-64-3], total NADH-cytochrome c reductase (EC 1.6.99.3) (IV) [9027-14-9], and cytochrome oxidase (EC 1.9.3.1) (V) [9001-16-5] activities in rat brain total and crude **mitochondrial** homogenates increased between the 16th and 20th wk of life and then decreased. (-)-Eburnamonine [474-00-0] increased **mitochondrial** V at all tested times; I increased and II decreased after treatment between the 16th and 24th wk, and also on treatment between the 24th and 28th wk only. Medibazine [53-31-6] increased **mitochondrial** III and V, effects disappearing after 12 wk, and also total homogenate III and IV. Trimetazidine [5011-34-7] increased **mitochondrial** II, III, and IV between the 16th and 20th wk. Papaverine [58-74-2] increased total homogenate I throughout and III and V between the 16th and 20th wk. Suloctidil [54767-75-8] increased total homogenate I, IV, and V and **mitochondrial** II and III between the 16th and 20th wk; increases in II were obsd. up to the 28th wk. Bamethan [3703-79-5] increased II, III, IV, and V after 8 wk treatment, and II and IV with treatment between the 24 and 28th wk only. Inositol niacinate [6556-11-2] increased total homogenate III only and UDP-glucose [**133-89-1**] showed no effects.

L7 ANSWER 48 OF 48 HCAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1972:414390 HCAPLUS
DOCUMENT NUMBER: 77:14390
TITLE: Motor nerve conduction velocity of normal and arteropathic subjects subjected to chronic **pharmacological** treatment with pyrimidine nucleosides
AUTHOR(S): Serra, C.
CORPORATE SOURCE: Serv. Neurofisiol. Clin., Cent. Traumatol. Ortop., Italy
SOURCE: Riforma Med. (1971), 85(50), 1544-51
CODEN: RIMEAB
DOCUMENT TYPE: Journal
LANGUAGE: Italian
AB Adults were given daily i.m. injections of 150 mg uridine (I) [**58-96-8**] plus 150 mg cytidine (II) [**65-46-3**] for 30 days. In normal subjects, no change in the elec. conduction velocity of the perineal or the posterior tibial nerve was noted, whereas an increase was seen in 15 of 30 patients with atherosclerosis and in 8 of 10 diabetics. The results are discussed in terms of the effect of I and II on nerve membrane potential and glucose uptake in atherosclerosis and on the vascular lesions underlying the **neuropathy** in diabetes.

=> select hit rn l7 1-48
E1 THROUGH E32 ASSIGNED

=> fil reg
FILE 'REGISTRY' ENTERED AT 16:08:02 ON 20 MAR 2002
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PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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STRUCTURE FILE UPDATES: 18 MAR 2002 HIGHEST RN 401788-64-5
DICTIONARY FILE UPDATES: 18 MAR 2002 HIGHEST RN 401788-64-5

TSCA INFORMATION NOW CURRENT THROUGH July 7, 2001

Please note that search-term pricing does apply when conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Calculated physical property data is now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

The P indicator for Preparations was not generated for all of the CAS Registry Numbers that were added to the H/Z/CA/CAplus files between 12/27/01 and 1/23/02. Use of the P indicator in online and SDI searches during this period, either directly appended to a CAS Registry Number or by qualifying an L-number with /P, may have yielded incomplete results. As of 1/23/02, the situation has been resolved. Also, note that searches

conducted using the PREP role indicator were not affected.

Customers running searches and/or SDIs in the H/Z/CA/CAplus files incorporating CAS Registry Numbers with the P indicator between 12/27/01 and 1/23/02, are encouraged to re-run these strategies. Contact the CAS Help Desk at 1-800-848-6533 in North America or 1-614-447-3698, worldwide, or send an e-mail to help@cas.org for further assistance or to receive a credit for any duplicate searches.

=> d his l8

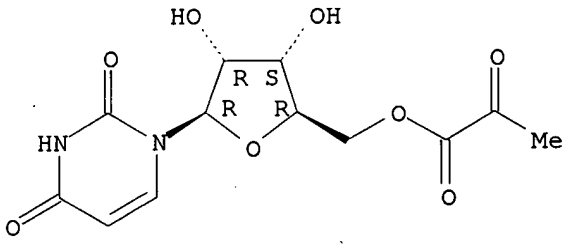
(FILE 'HCAPLUS' ENTERED AT 16:05:35 ON 20 MAR 2002)
SELECT HIT RN L7 1-48

FILE 'REGISTRY' ENTERED AT 16:08:02 ON 20 MAR 2002
L8 32 S E1-E32

=> d ide can l8 1-32

L8 ANSWER 1 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **260360-07-4** REGISTRY
CN Uridine, 5'-(2-oxopropanoate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C12 H14 N2 O8
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



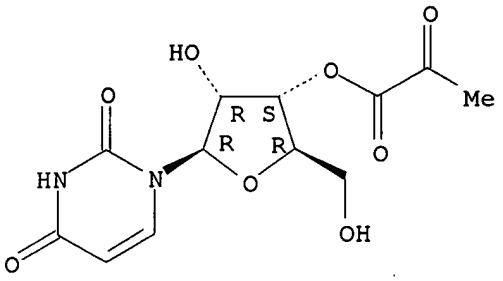
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 132:203149

L8 ANSWER 2 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **260360-06-3** REGISTRY
CN Uridine, 3'-(2-oxopropanoate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C12 H14 N2 O8
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



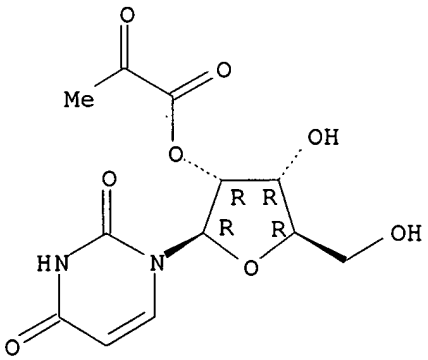
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 132:203149

L8 ANSWER 3 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **260360-05-2** REGISTRY
CN Uridine, 2'-(2-oxopropanoate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C12 H14 N2 O8
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



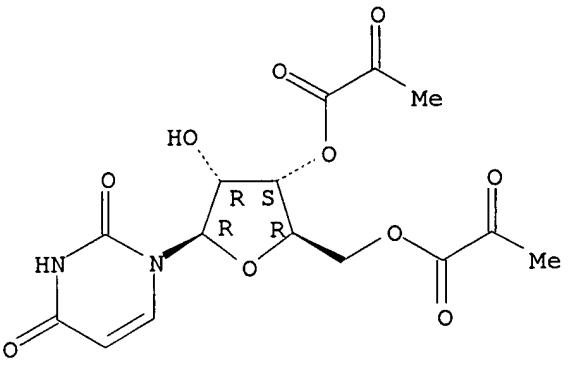
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 132:203149

L8 ANSWER 4 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **260360-04-1** REGISTRY
CN Uridine, 3',5'-bis(2-oxopropanoate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C15 H16 N2 O10
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



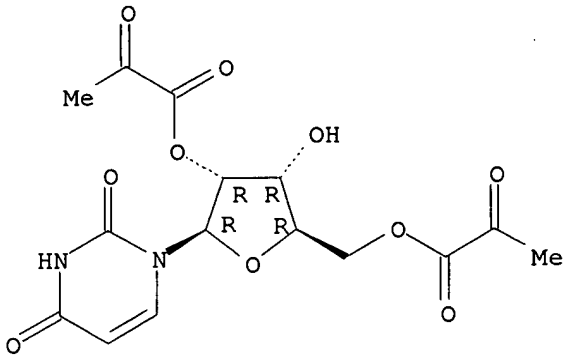
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 132:203149

L8 ANSWER 5 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **260360-03-0** REGISTRY
CN Uridine, 2',5'-bis(2-oxopropanoate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C15 H16 N2 O10
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



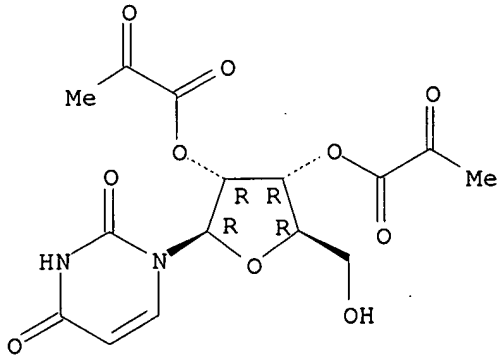
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 132:203149

L8 ANSWER 6 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **260360-02-9** REGISTRY
CN Uridine, 2',3'-bis(2-oxopropanoate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C15 H16 N2 O10
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



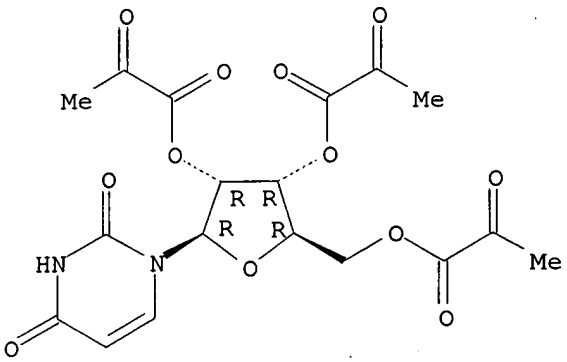
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 132:203149

L8 ANSWER 7 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **260360-01-8** REGISTRY
CN Uridine, 2',3',5'-tris(2-oxopropanoate) (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C18 H18 N2 O12
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



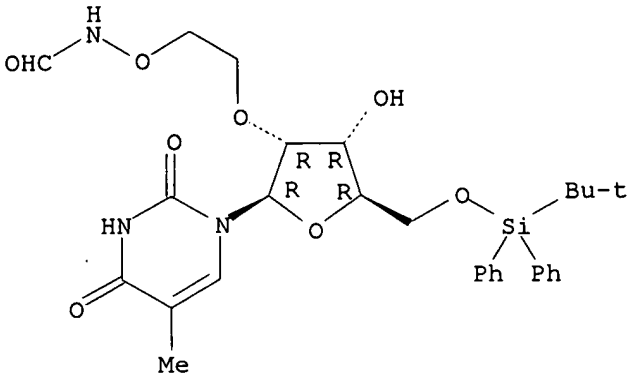
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 132:203149

L8 ANSWER 8 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **244277-62-1** REGISTRY
CN Uridine, 5'-O-[(1,1-dimethylethyl)diphenylsilyl]-2'-O-[2-[(formylamino)oxy]ethyl]-5-methyl- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C29 H37 N3 O8 Si
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



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REFERENCE 2: 134:353482

REFERENCE 3: 133:276321

REFERENCE 4: 132:231981

REFERENCE 5: 132:231980

REFERENCE 6: 132:203170

REFERENCE 7: 132:203163

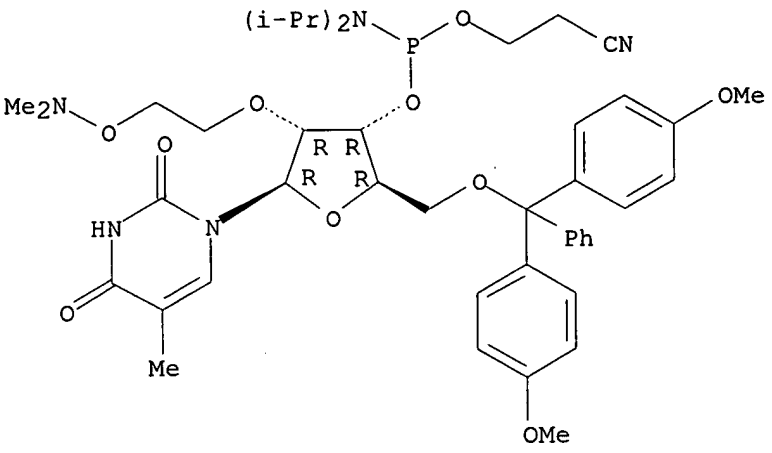
REFERENCE 8: 132:132354

REFERENCE 9: 131:252587

REFERENCE 10: 131:252586

L8 ANSWER 9 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **212061-30-8** REGISTRY
CN Uridine, 5'-O-[bis(4-methoxyphenyl)phenylmethyl]-2'-O-[2-[(dimethylamino)oxy]ethyl]-5-methyl-, 3'-[2-cyanoethyl bis(1-methylethyl)phosphoramidite] (9CI) (CA INDEX NAME)
FS STEREOSEARCH
DR 249764-68-9
MF C44 H58 N5 O10 P
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

68 REFERENCES IN FILE CA (1967 TO DATE)
69 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 136:178013

REFERENCE 2: 136:112689

REFERENCE 3: 136:37881

REFERENCE 4: 135:339219

REFERENCE 5: 135:283219

REFERENCE 6: 135:236463

REFERENCE 7: 135:147408

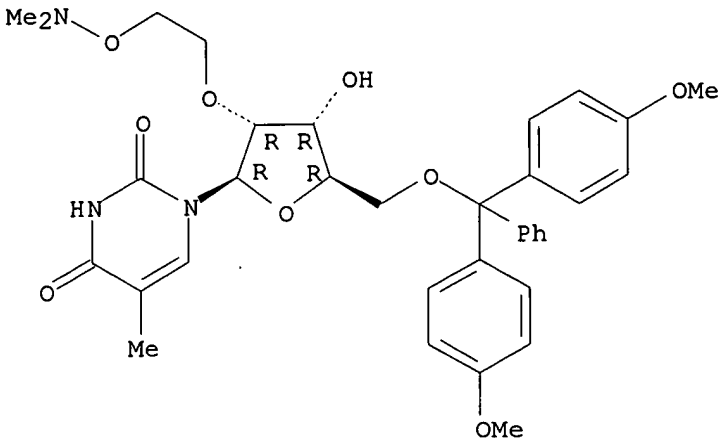
REFERENCE 8: 135:132466

REFERENCE 9: 135:132465

REFERENCE 10: 135:132463

L8 ANSWER 10 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **212061-29-5** REGISTRY
CN Uridine, 5'-O-[bis(4-methoxyphenyl)phenylmethyl]-2'-O-[2-[(dimethylamino)oxy]ethyl]-5-methyl- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C35 H41 N3 O9
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

69 REFERENCES IN FILE CA (1967 TO DATE)
70 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 136:178013

REFERENCE 2: 136:112689

REFERENCE 3: 135:339219

REFERENCE 4: 135:283223

REFERENCE 5: 135:283219

REFERENCE 6: 135:236463

REFERENCE 7: 135:147408

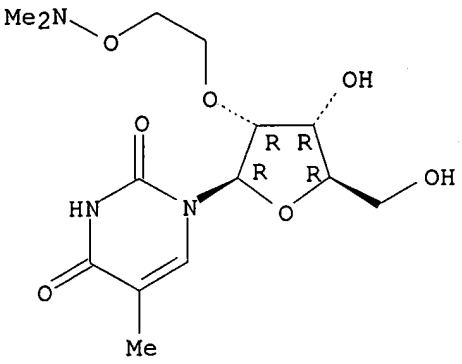
REFERENCE 8: 135:132466

REFERENCE 9: 135:132465

REFERENCE 10: 135:132463

L8 ANSWER 11 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **212061-28-4** REGISTRY
CN Uridine, 2'-O-[2-[(dimethylamino)oxy]ethyl]-5-methyl- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
MF C14 H23 N3 O7
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

70 REFERENCES IN FILE CA (1967 TO DATE)
71 REFERENCES IN FILE CAPLUS (1967 TO DATE)

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REFERENCE 2: 136:112689

REFERENCE 3: 135:339219

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REFERENCE 5: 135:283219

REFERENCE 6: 135:236463

REFERENCE 7: 135:147408

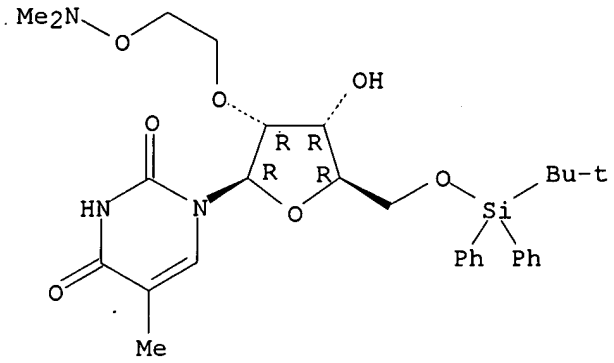
REFERENCE 8: 135:132466

REFERENCE 9: 135:132465

REFERENCE 10: 135:132463

L8 ANSWER 12 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **212061-27-3** REGISTRY
CN Uridine, 2'-O-[2-[(dimethylamino)oxy]ethyl]-5'-O-[(1,1-dimethylethyl)diphenylsilyl]-5-methyl- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
DR 244121-68-4
MF C30 H41 N3 O7 Si
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

71 REFERENCES IN FILE CA (1967 TO DATE)
72 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 136:178013

REFERENCE 2: 136:112689

REFERENCE 3: 135:366770

REFERENCE 4: 135:339219

REFERENCE 5: 135:283223

REFERENCE 6: 135:283219

REFERENCE 7: 135:236463

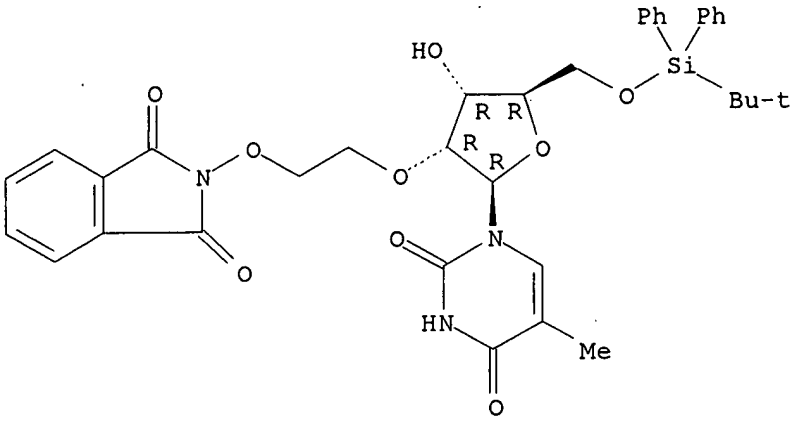
REFERENCE 8: 135:147408

REFERENCE 9: 135:132466

REFERENCE 10: 135:132465

L8 ANSWER 13 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **212061-25-1** REGISTRY
CN Uridine, 2'-O-[2-[(1,3-dihydro-1,3-dioxo-2H-isoindol-2-yl)oxy]ethyl]-5'-O-[(1,1-dimethylethyl)diphenylsilyl]-5-methyl- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
DR 244121-66-2, 249764-66-7
MF C36 H39 N3 O9 Si
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



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REFERENCE 2: 136:112689

REFERENCE 3: 135:366770

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REFERENCE 7: 135:236463

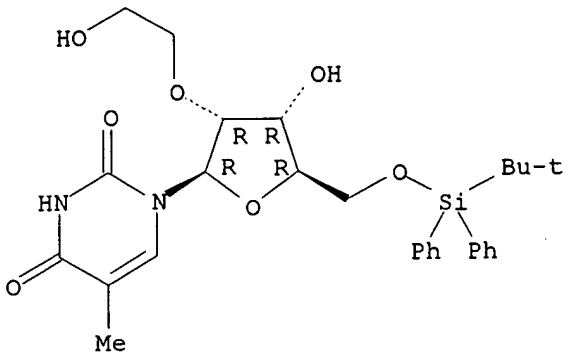
REFERENCE 8: 135:147408

REFERENCE 9: 135:132466

REFERENCE 10: 135:132465

L8 ANSWER 14 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **212061-24-0** REGISTRY
CN Uridine, 5'-O-[(1,1-dimethylethyl)diphenylsilyl]-2'-O-(2-hydroxyethyl)-5-methyl- (9CI) (CA INDEX NAME)
FS STEREOSEARCH
DR 244121-65-1
MF C28 H36 N2 O7 Si
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

71 REFERENCES IN FILE CA (1967 TO DATE)
72 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 136:178013

REFERENCE 2: 136:112689

REFERENCE 3: 135:366770

REFERENCE 4: 135:339219

REFERENCE 5: 135:283223

REFERENCE 6: 135:283219

REFERENCE 7: 135:236463

REFERENCE 8: 135:147408

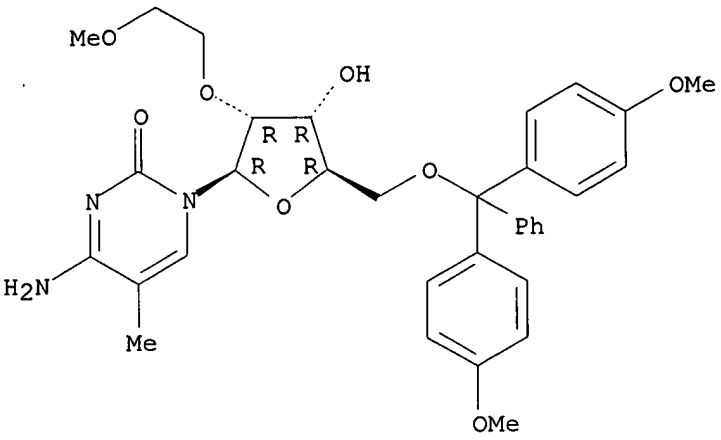
REFERENCE 9: 135:132466

REFERENCE 10: 135:132465

L8 ANSWER 15 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **182496-00-0** REGISTRY
CN Cytidine, 5'-O-[bis(4-methoxyphenyl)phenylmethyl]-2'-O-(2-methoxyethyl)-5-methyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 11: PN: WO0018781 PAGE: 35 claimed sequence
CN 11: PN: WO0020645 PAGE: 34 claimed sequence
CN 2'-O-Methoxyethyl-5-O-dimethoxytrityl-5-methylcytidine
CN 24: PN: US6004814 PAGE: 19 claimed sequence
FS STEREOSEARCH
MF C34 H39 N3 O8
SR CA

LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



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REFERENCE 7: 135:236463

REFERENCE 8: 135:175354

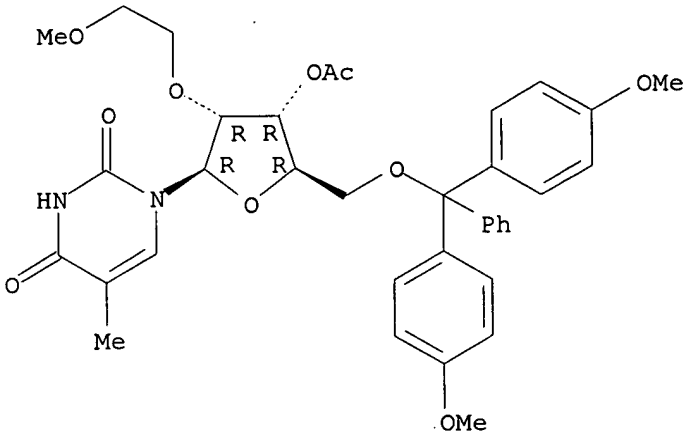
REFERENCE 9: 135:147408

REFERENCE 10: 135:132466

L8 ANSWER 16 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **182495-98-3** REGISTRY
CN Uridine, 5'-O-[bis(4-methoxyphenyl)phenylmethyl]-2'-O-(2-methoxyethyl)-5-methyl-, 3'-acetate (9CI) (CA INDEX NAME)
OTHER NAMES:

CN 19: PN: US6004814 PAGE: 18 claimed sequence
CN 3'-O-Acetyl-2'-O-methoxyethyl-5-O-dimethoxytrityl-5-methyluridine
CN 7: PN: WO0018781 PAGE: 34 claimed sequence
CN 7: PN: WO0020645 PAGE: 33 claimed sequence
FS STEREOSEARCH
MF C36 H40 N2 O10
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

127 REFERENCES IN FILE CA (1967 TO DATE)
10 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
127 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 136:178013

REFERENCE 2: 136:112689

REFERENCE 3: 136:79745

REFERENCE 4: 135:366770

REFERENCE 5: 135:339219

REFERENCE 6: 135:283223

REFERENCE 7: 135:283219

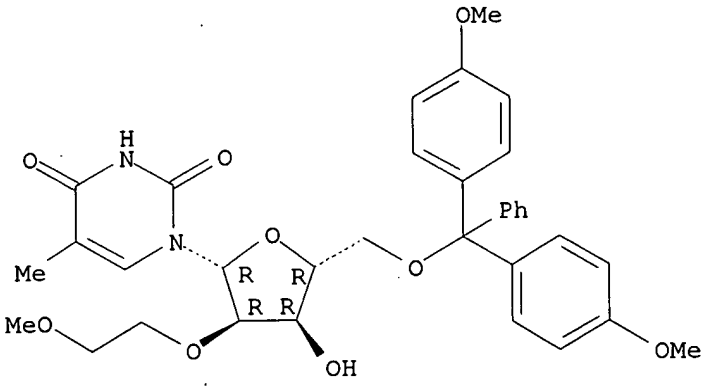
REFERENCE 8: 135:236463

REFERENCE 9: 135:175354

REFERENCE 10: 135:147408

L8 ANSWER 17 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **163759-50-0** REGISTRY
CN Uridine, 5'-O-[bis(4-methoxyphenyl)phenylmethyl]-2'-O-(2-methoxyethyl)-5-methyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2'-O-Methoxyethyl-5-O-dimethoxytrityl-5-methyluridine
CN 20: PN: US6004814 PAGE: 18 claimed sequence
CN 5: PN: WO0018781 PAGE: 33 claimed sequence
CN 5: PN: WO0020645 PAGE: 33 claimed sequence
FS STEREOSEARCH
MF C34 H38 N2 O9
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

138 REFERENCES IN FILE CA (1967 TO DATE)
138 REFERENCES IN FILE CAPLUS (1967 TO DATE)

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REFERENCE 3: 136:86027

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REFERENCE 5: 136:79745

REFERENCE 6: 135:366770

REFERENCE 7: 135:339219

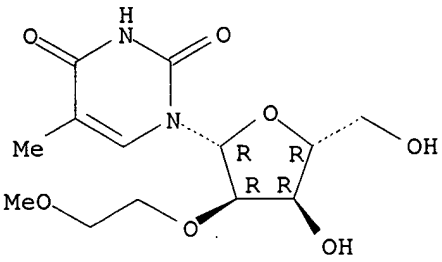
REFERENCE 8: 135:283223

REFERENCE 9: 135:283219

REFERENCE 10: 135:236463

L8 ANSWER 18 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **163759-49-7** REGISTRY
CN Uridine, 2'-O-(2-methoxyethyl)-5-methyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2'-O-Methoxyethyl-5-methyluridine
CN 3: PN: WO0018781 PAGE: 32 claimed sequence
CN 3: PN: WO0020645 PAGE: 32 claimed sequence
FS STEREOSEARCH
MF C13 H20 N2 O7
SR CA
LC STN Files: CA, CAPLUS, TOXCENTER, USPATFULL

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

129 REFERENCES IN FILE CA (1967 TO DATE)
129 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 136:178013

REFERENCE 2: 136:112689

REFERENCE 3: 136:79745

REFERENCE 4: 135:366770

REFERENCE 5: 135:339219

REFERENCE 6: 135:283223

REFERENCE 7: 135:283219

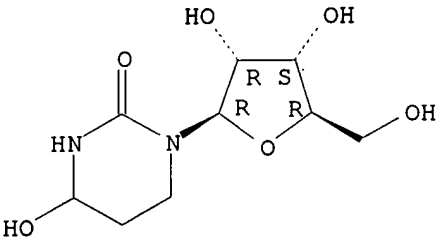
REFERENCE 8: 135:236463

REFERENCE 9: 135:175354

REFERENCE 10: 135:147408

L8 ANSWER 19 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **18771-50-1** REGISTRY
CN 2(1H)-Pyrimidinone, tetrahydro-4-hydroxy-1-.beta.-D-ribofuranosyl- (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1-(.beta.-D-Ribofuranosyl)-4-hydroxytetrahydro-1(1H)-pyrimidinone
CN 3,4,5,6-Tetrahydrouridine
CN NSC 112907
CN Tetrahydrouridine
FS STEREOSEARCH
DR 68060-67-3
MF C9 H16 N2 O6
CI COM
LC STN Files: BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CHEMCATS, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, PROMT, RTECS*, SYNTHLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

93 REFERENCES IN FILE CA (1967 TO DATE)
93 REFERENCES IN FILE CAPLUS (1967 TO DATE)

REFERENCE 1: 135:266684

REFERENCE 2: 135:146758

REFERENCE 3: 134:349905

REFERENCE 4: 133:219596

REFERENCE 5: 131:319566

REFERENCE 6: 131:281604

REFERENCE 7: 130:278417

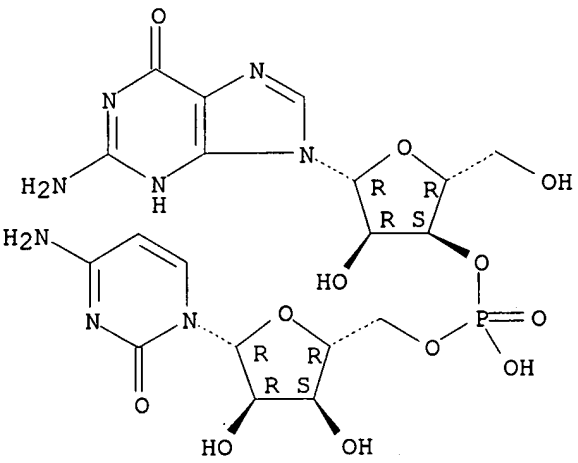
REFERENCE 8: 126:139905 - No

REFERENCE 9: 125:284875

REFERENCE 10: 123:187874

L8 ANSWER 20 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **4785-04-0** REGISTRY
CN Cytidine, guanylyl-(3'.fwdarw.5')- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 3'-Guanylic acid, 5'-ester with cytidine (6CI)
CN Guanosine, cytidylyl-(5'.fwdarw.3')- (7CI, 8CI)
OTHER NAMES:
CN Guanylyl-(3',5')-cytidine
FS STEREOSEARCH
MF C19 H25 N8 O12 P
CI COM
LC STN Files: AGRICOLA, BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMLIST, CSCHEM, MEDLINE, TOXCENTER
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

273 REFERENCES IN FILE CA (1967 TO DATE)
7 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
273 REFERENCES IN FILE CAPLUS (1967 TO DATE)
19 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:164502

REFERENCE 2: 136:164348

REFERENCE 3: 136:3325

REFERENCE 4: 135:314971

REFERENCE 5: 135:253549

REFERENCE 6: 134:51114

REFERENCE 7: 134:29645

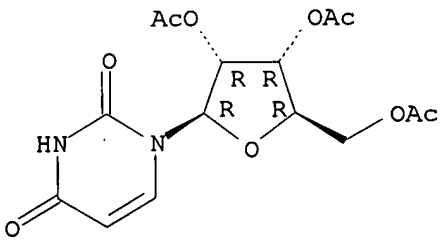
REFERENCE 8: 133:238236

REFERENCE 9: 133:115747

REFERENCE 10: 133:13811

L8 ANSWER 21 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **4105-38-8** REGISTRY
CN Uridine, 2',3',5'-triacetate (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2',3',5'-Tri-O-acetyluridine
CN 2',3',5'-Triacetyluridine
CN Tri-O-acetyl uridine
FS STEREOSEARCH
DR 293738-13-3
MF C15 H18 N2 O9
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DRUGUPDATES, HODOC*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

161 REFERENCES IN FILE CA (1967 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
161 REFERENCES IN FILE CAPLUS (1967 TO DATE)

4 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:69462

REFERENCE 2: 135:371938

REFERENCE 3: 135:358112

REFERENCE 4: 135:358102

REFERENCE 5: 135:191326

REFERENCE 6: 134:260942

REFERENCE 7: 134:71820

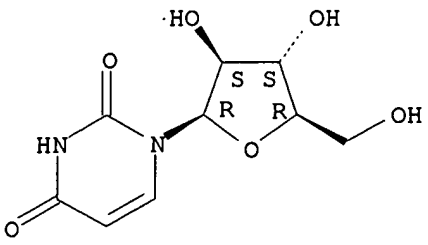
REFERENCE 8: 133:238227

REFERENCE 9: 133:187987

REFERENCE 10: 133:144540

L8 ANSWER 22 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **3083-77-0** REGISTRY
CN 2,4(1H,3H)-Pyrimidinedione, 1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Uracil, 1-.beta.-D-arabinofuranosyl- (6CI, 7CI, 8CI)
OTHER NAMES:
CN 1-.beta.-D-Arabinofuranosyluracil
CN Ara-U
CN Arabinofuranosyluracil
CN Arabinosyluracil
CN Arauridine
CN Spongouridin
CN Spongouridine
CN Uracil .beta.-D-arabinofuranoside
CN Uracil arabinoside
FS STEREOSEARCH
DR 489-61-2, 92418-86-5
MF C9 H12 N2 O6
CI COM
LC STN Files: ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DDFU, DRUGU, EMBASE, GMELIN*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, NAPRALERT, PROMT, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

340 REFERENCES IN FILE CA (1967 TO DATE)
14 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
342 REFERENCES IN FILE CAPLUS (1967 TO DATE)
14 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:37854

REFERENCE 2: 135:338694

REFERENCE 3: 135:81920

REFERENCE 4: 135:55373

REFERENCE 5: 134:260835

REFERENCE 6: 134:246868

REFERENCE 7: 134:125521

REFERENCE 8: 133:246786

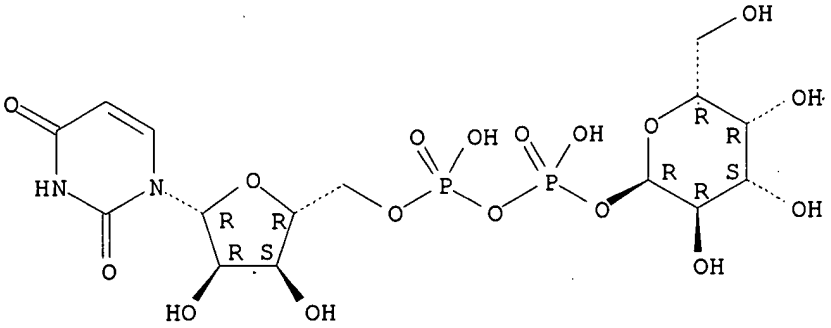
REFERENCE 9: 133:88295

REFERENCE 10: 132:329490

L8 ANSWER 23 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **2956-16-3** REGISTRY
CN Uridine 5'-(trihydrogen diphosphate), P'-.alpha.-D-galactopyranosyl ester (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Uridine 5'-(trihydrogen pyrophosphate), mono-.alpha.-D-galactopyranosyl ester (8CI)
CN Uridine 5'-pyrophosphate, .alpha.-D-galactopyranosyl ester (6CI, 7CI)
OTHER NAMES:
CN UDP-.alpha.-D-Galactose
CN UDP-D-galactose
CN UDP-galactopyranose
CN UDP-galactose
CN Uridine 5'-(.alpha.-D-galactopyranosyl pyrophosphate)
CN Uridine 5'-diphosphate galactose
CN Uridine 5'-diphosphogalactose

CN Uridine 5'-pyrophosphate, .alpha.-D-galactosyl ester
CN Uridine 5'-pyrophosphate, D-galactosyl ester
CN Uridine diphosphate-D-galactose
CN Uridine pyrophosphate, .alpha.-D-galactopyranosyl ester
CN Uridinediphosphate galactose
CN Uridinediphosphogalactose
FS STEREOSEARCH
DR 17012-87-2, 98242-76-3, 99005-44-4, 99094-94-7, 4220-91-1, 27234-73-7, 30138-01-3, 99945-86-5
MF C15 H24 N2 O17 P2
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMINFORMRX, CSCHEM, EMBASE, IFICDB, IFIPAT, IFIUDB, MEDLINE, NAPRALERT, PROMT, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

868 REFERENCES IN FILE CA (1967 TO DATE)
9 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
871 REFERENCES IN FILE CAPLUS (1967 TO DATE)
23 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:182545

REFERENCE 2: 136:180398

REFERENCE 3: 136:180268

REFERENCE 4: 136:145922

REFERENCE 5: 136:129727

REFERENCE 6: 136:118653

REFERENCE 7: 136:114649

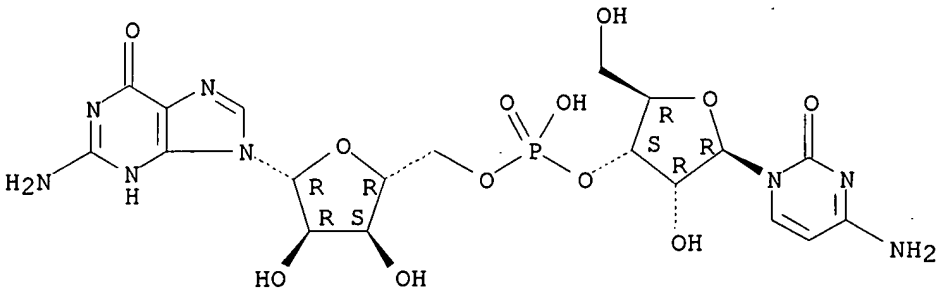
REFERENCE 8: 136:114425

REFERENCE 9: 136:66096

REFERENCE 10: 136:50169

L8 ANSWER 24 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **2382-65-2** REGISTRY
CN Guanosine, cytidylyl-(3'.fwdarw.5')- (7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN (3'-5')CpG
CN Cytidine, guanylyl-(5'.fwdarw.3')-
CN Cytidylyl-(3',5')-guanosine
CN Cytidylylguanosine
CN Guanosine cytidine 3',5'-monophosphate
FS STEREOSEARCH
DR 122138-10-7, 72507-03-0
MF C19 H25 N8 O12 P
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CANCERLIT, CAOLD, CAPLUS, CHEMCATS, CHEMLIST, EMBASE, MEDLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

390 REFERENCES IN FILE CA (1967 TO DATE)
30 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
392 REFERENCES IN FILE CAPLUS (1967 TO DATE)
12 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:182447

REFERENCE 2: 136:182440

REFERENCE 3: 136:182299

REFERENCE 4: 136:166156

REFERENCE 5: 136:162287

REFERENCE 6: 136:133605

REFERENCE 7: 136:133208

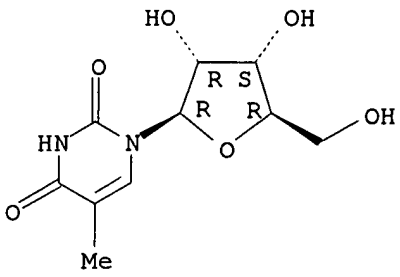
REFERENCE 8: 136:131323

REFERENCE 9: 136:116076

REFERENCE 10: 136:114387

L8 ANSWER 25 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **1463-10-1** REGISTRY
CN Uridine, 5-methyl- (6CI, 7CI, 8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN .beta.-D-Ribofuranoside, thymine-1
CN 1-.beta.-D-Ribofuranosylthymine
CN 16: PN: US6004814 PAGE: 17 claimed sequence
CN 2,4(1H,3H)-Pyrimidinedione, 5-methyl-1-.beta.-D-ribofuranosyl-
CN 2: PN: WO0018781 PAGE: 32 claimed sequence
CN 2: PN: WO0020645 PAGE: 32 claimed sequence
CN 5-Methyluridine
CN PN: WO9947707 PAGE: 62-66 claimed sequence
CN Ribothymidine
CN Thymine riboside
FS STEREOSEARCH
MF C10 H14 N2 O6
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DDFU, DRUGU, EMBASE, MEDLINE, MSDS-OHS, PROMT, SPECINFO, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

559 REFERENCES IN FILE CA (1967 TO DATE)
10 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
560 REFERENCES IN FILE CAPLUS (1967 TO DATE)
22 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:178013

REFERENCE 2: 136:147620

REFERENCE 3: 136:112689

REFERENCE 4: 136:81435

REFERENCE 5: 136:79745

REFERENCE 6: 135:339219

REFERENCE 7: 135:283223

REFERENCE 8: 135:283219

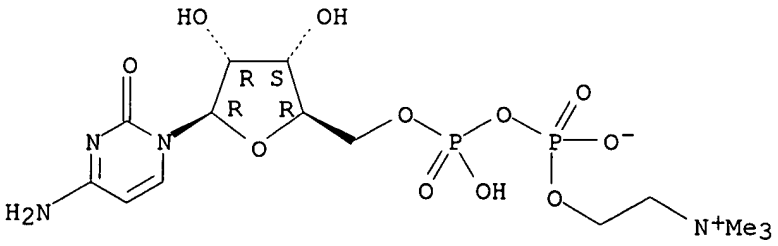
REFERENCE 9: 135:242463

REFERENCE 10: 135:236463

L8 ANSWER 26 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **987-78-0** REGISTRY
CN Cytidine 5'-(trihydrogen diphosphate), P'-[2-(trimethylammonio)ethyl] ester, inner salt (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Choline, hydroxide, 5'-ester with cytidine 5'-(trihydrogen pyrophosphate), inner salt (8CI)
CN Cytidine 5'-(trihydrogen diphosphate), P'-[2-(trimethylammonio)ethyl] ester, hydroxide, inner salt
OTHER NAMES:
CN CDP-choline
CN Cereb
CN Choline 5'-cytidine diphosphate

CN Choline cytidine diphosphate
CN Citicholine
CN Citicoline
CN Citidoline
CN Colite
CN Cytidine 5'-(choline diphosphate)
CN Cytidine 5'-(cholinyl pyrophosphate)
CN Cytidine 5'-diphosphate choline
CN Cytidine 5'-diphosphocholine
CN Cytidine choline diphosphate
CN Cytidine diphosphate choline
CN Cytidine diphosphate choline ester
CN Cytidine diphosphocholine
CN Cytidine diphosphorylcholine
CN Cytidoline
CN Ensign
CN Nicholin
CN Nicolin
CN Niticolin
CN Recofnan
CN Recognan
CN Somazina
CN Suncholin
FS STEREOSEARCH
DR 1477-47-0, 64143-42-6
MF C14 H26 N4 O11 P2
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGNL, DRUGU, DRUGUPDATES, EMBASE, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PHAR, PHARMASEARCH, PROMT, RTECS*, TOXCENTER, USAN, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



719 REFERENCES IN FILE CA (1967 TO DATE)
10 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
720 REFERENCES IN FILE CAPLUS (1967 TO DATE)
5 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:160832

REFERENCE 2: 136:128931

REFERENCE 3: 136:128907

REFERENCE 4: 136:96534

REFERENCE 5: 136:79648

REFERENCE 6: 136:50169

REFERENCE 7: 135:268687

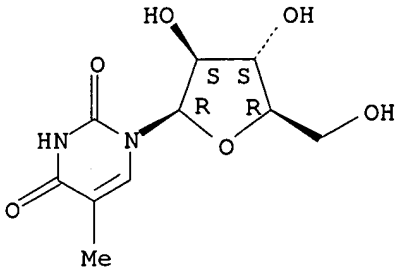
REFERENCE 8: 135:251959

REFERENCE 9: 135:236455

REFERENCE 10: 135:170846

L8 ANSWER 27 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **605-23-2** REGISTRY
CN 2,4(1H,3H)-Pyrimidinedione, 1-.beta.-D-arabinofuranosyl-5-methyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Thymine, 1-.beta.-D-arabinofuranosyl- (6CI, 7CI, 8CI)
OTHER NAMES:
CN 1-.beta.-D-Arabinofuranosylthymine
CN 5-Methylarabinosyluracil
CN Ara-T
CN Arabinosylthymine
CN Spongothymidin
CN Spongothymidine
CN Thymine arabinoside
FS STEREOSEARCH
DR 2946-29-4
MF C10 H14 N2 O6
CI COM
LC STN Files: AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DDFU, DRUGU, EMBASE, MEDLINE, NAPRALERT, PROMT, RTECS*, TOXCENTER, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

202 REFERENCES IN FILE CA (1967 TO DATE)
6 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
202 REFERENCES IN FILE CAPLUS (1967 TO DATE)
15 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:160968

REFERENCE 2: 136:129036

REFERENCE 3: 136:17266

REFERENCE 4: 135:340189

REFERENCE 5: 135:338800

REFERENCE 6: 135:163726

REFERENCE 7: 135:137667

REFERENCE 8: 135:71226

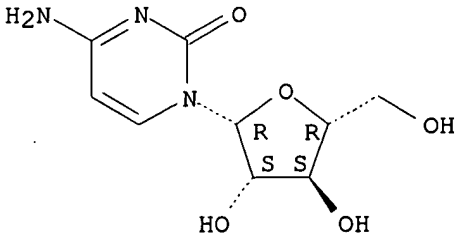
REFERENCE 9: 135:70775

REFERENCE 10: 135:1220

L8 ANSWER 28 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **147-94-4** REGISTRY
CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-arabinofuranosyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Cytosine, 1-.beta.-D-arabinofuranosyl- (6CI, 8CI)
OTHER NAMES:
CN (Arabinofuranosyl)cytosine
CN 1-(.beta.-D-Arabinofuranosyl)cytosine
CN 1-(Arabinofuranosyl)cytosine
CN 1-.beta.-Arabinofuranosylcytosine
CN 1-.beta.-D-Arabinosylcytosine
CN 4-Amino-1-arabinofuranosyl-2-oxo-1,2-dihydropyrimidine

CN 58: PN: US6159940 SEQID: 71 claimed sequence
CN Ac 1075
CN Alexan
CN Ara-C
CN ara-Cytosine
CN Arabinocytosine
CN Arabinoside C
CN Aracytidine
CN Aracytin
CN Aracytine
CN Arafcyt
CN Citozar
CN Cyclocide
CN Cytarabin
CN Cytarabine
CN Cytarabinoside
CN Cytosar
CN Cytosine .beta.-D-arabinofuranoside
CN Cytosine .beta.-D-arabinoside
CN Cytosine arabinoside
CN Cytosine-1-.beta.-arabinofuranoside
CN Cytosine-1-.beta.-D-arabinofuranoside
CN DepoCyte
CN NSC 63878
CN Spongocytidine
CN U 19920
CN U 19920A
CN Udicil
FS STEREOSEARCH
MF C9 H13 N3 O5
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGU, EMBASE, GMELIN*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PHAR, PHARMASEARCH, PROMT, RTECS*, TOXCENTER, USAN, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

5002 REFERENCES IN FILE CA (1967 TO DATE)
140 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
5014 REFERENCES IN FILE CAPLUS (1967 TO DATE)
30 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:179006

REFERENCE 2: 136:177694

REFERENCE 3: 136:177616

REFERENCE 4: 136:177585

REFERENCE 5: 136:177559

REFERENCE 6: 136:172758

REFERENCE 7: 136:172755

REFERENCE 8: 136:166066

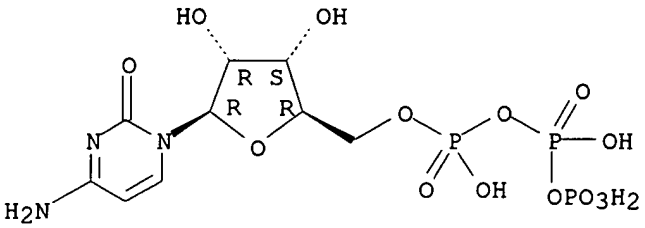
REFERENCE 9: 136:164610

REFERENCE 10: 136:161339

All - 1999

L8 ANSWER 29 OF 32 REGISTRY COPYRIGHT 2002 ACS
RN **65-47-4** REGISTRY
CN Cytidine 5'-(tetrahydrogen triphosphate) (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 5'-CTP
CN CTP
CN Cytidine 5'-triphosphate
CN Cytidine triphosphate
CN Cytidine, mono(tetrahydrogen triphosphate) (ester)
FS STEREOSEARCH
MF C9 H16 N3 O14 P3
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMLIST, CSCHEM, DDFU, DRUGU, EMBASE, IPA, MEDLINE, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

2518 REFERENCES IN FILE CA (1967 TO DATE)
55 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2520 REFERENCES IN FILE CAPLUS (1967 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:184057

REFERENCE 2: 136:163900

REFERENCE 3: 136:162375

REFERENCE 4: 136:162032

REFERENCE 5: 136:145691

REFERENCE 6: 136:115505

REFERENCE 7: 136:114697

REFERENCE 8: 136:100194

REFERENCE 9: 136:95680

REFERENCE 10: 136:66042

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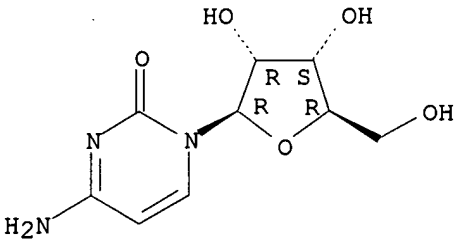
RN **65-46-3** REGISTRY
CN Cytidine (8CI, 9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Cytosine, 1-.beta.-D-ribosyl- (6CI)
OTHER NAMES:
CN .beta.-D-Ribofuranoside, cytosine-1
CN 1-(.beta.-D-Ribofuranosyl)-2-oxo-4-amino-1,2-dihydro-1,3-diazine
CN 1-.beta.-D-Ribofuranosylcytosine
CN 2(1H)-Pyrimidinone, 4-amino-1-.beta.-D-ribofuranosyl-
CN 4-Amino-1-.beta.-D-ribofuranosyl-2(1H)-pyrimidinone
CN Cytosine riboside
FS STEREOSEARCH
DR 4395-95-3
MF C9 H13 N3 O5

CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DETHERM*, DRUGU, EMBASE, GMELIN*, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3138 REFERENCES IN FILE CA (1967 TO DATE)
192 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
3141 REFERENCES IN FILE CAPLUS (1967 TO DATE)
50 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:177961

REFERENCE 2: 136:163471

REFERENCE 3: 136:162375

REFERENCE 4: 136:151382

REFERENCE 5: 136:147251

REFERENCE 6: 136:114391

REFERENCE 7: 136:102614

REFERENCE 8: 136:102226

REFERENCE 9: 136:102057

REFERENCE 10: 136:101233

All > 1999

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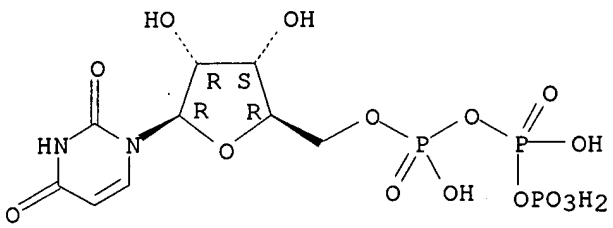
RN **63-39-8** REGISTRY
CN Uridine 5'-(tetrahydrogen triphosphate) (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 5'-UTP
CN Uridine 5'-triphosphate
CN Uridine triphosphate
CN Uridine, mono(tetrahydrogen triphosphate) (ester)
CN Uteplex
CN UTP
FS STEREOSEARCH
MF C9 H15 N2 O15 P3
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CEN, CHEMLIST, CIN, CSCHEM, DDFU, DRUGNL, DRUGU, DRUGUPDATES, EMBASE, GMELIN*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, NIOSHTIC, PROMT, RTECS*, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

3523 REFERENCES IN FILE CA (1967 TO DATE)
73 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
3526 REFERENCES IN FILE CAPLUS (1967 TO DATE)
9 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:179424

REFERENCE 2: 136:178256

REFERENCE 3: 136:178223

REFERENCE 4: 136:164777

REFERENCE 5: 136:163900

REFERENCE 6: 136:162375

REFERENCE 7: 136:162335

REFERENCE 8: 136:151393

REFERENCE 9: 136:145541

REFERENCE 10: 136:132037

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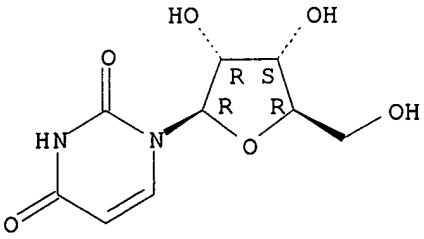
RN **58-96-8** REGISTRY
CN Uridine (8CI, 9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Uracil, 1-.beta.-D-ribofuranosyl- (7CI)
OTHER NAMES:
CN .beta.-D-Ribofuranoside, 2,4(1H,3H)-pyrimidinedione-1
CN .beta.-Uridine
CN 1-.beta.-D-Ribofuranosyl-2,4(1H,3H)-pyrimidinedione
CN 1-.beta.-D-Ribofuranosyluracil
CN Uridin
FS STEREOSEARCH
DR 12693-39-9, 68184-15-6
MF C9 H12 N2 O6
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DETHERM*, DRUGU, EMBASE, GMELIN*, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PHAR, PIRA, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

5115 REFERENCES IN FILE CA (1967 TO DATE)
322 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
5119 REFERENCES IN FILE CAPLUS (1967 TO DATE)

1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 136:184059

REFERENCE 2: 136:184040

REFERENCE 3: 136:179090

REFERENCE 4: 136:178283

REFERENCE 5: 136:163471

REFERENCE 6: 136:162375

REFERENCE 7: 136:150755

REFERENCE 8: 136:147251

REFERENCE 9: 136:147146

REFERENCE 10: 136:146690